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PULMONARY

TUBERCULOSIS

BY

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THIRD EDITION, REVISED AND ENLARGED

ILLUSTRATED WITH 129 ENGRAVINGS AND 28 PLATES

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TO
MY WIFE

PREFACE TO THE THIRD EDITION.

THE cordial reception accorded this book, of which two editions have been exhausted within five years, has offered an opportunity for radical changes in this new edition with a view of making it a more useful guide for the general practitioner as well as the specialist. Two new chapters have been introduced: One on the Reciprocal Relations Between Pulmonary Tuberculosis and Certain Physiological and Pathological Processes, and another on the Medical-legal and Insurance Aspects of Tuberculosis. In the former, a detailed discussion of the following topics is offered: The reciprocal influences exercised by tuberculosis and such physiological processes as growth, puberty, sexual functions, fecundation, pregnancy, labor, the puerperium, etc. Also a consideration of the coexistence of pulmonary tuberculosis with such pathological processes as extra-thoracic tuberculous lesions, diseases of the upper respiratory tract, especially tonsillitis, coryza, and the lymphatic diathesis; diseases of the bronchi and lung, notably asthma, bronchitis, bronchiectasis, emphysema, pneumonia, etc.; constitutional and metabolic diseases, such as the various forms of rheumatism, gout, diabetes, cholelithiasis, nephrolithiasis, obesity, and dysfunctions of the endocrine glands, the thyroid, the adrenals and the gonads; infectious diseases, notably the exanthemata, influenza, typhoid fever, syphilis, etc.; diseases of the gastrointestinal tract, appendicitis, fistula-in-ano, cirrhosis of the liver, etc.; and finally, cancer and insanity. It has been borne in mind that phthisical patients often live for many years, and that during their period of illness they are liable to suffer from various other diseases; and therefore the reciprocal relations discussed in this chapter have more than theoretical bearing on the symptomatology, course and prognosis. As far as the writer knows, this is the first attempt to treat this subject in any book or monograph on tuberculosis.

The new chapter on the Medico-legal and Insurance Aspects of Tuberculosis has been included because of the immense interest recently displayed by life and health insurance corporations, fraternal organ-

izations, labor unions, workmen's compensation commissions, veteran rehabilitation boards, etc., in the problems of the diagnosis of tuberculosis and the disability it causes. Traumatic tuberculosis has of late very often been considered in courts of law in claims for damages, the contention being that injuries were responsible for the origin or aggravation of tuberculous lung lesions.

The fact that the publishers consented to reset the entire book has made it possible to make very radical changes in nearly all the chapters. Those treating of Phthisiogenesis were rewritten, and emphasis has been laid on the problems of the constitutional *vs.* environmental factors in the etiology of tuberculous diseases, because most authorities are now in agreement that infection with tubercle bacilli does not alone explain fully the origin of phthisis in any of its forms. The chapter on artificial pneumothorax has been rewritten and amplified in the light of more extended experience, special emphasis being laid on the limitations of this mode of treatment. Many new sections have been included, the most important being on acute miliary tuberculosis, scrofula, myoidema, the autourine test, phototherapy, artificial immunization, etc. Many new illustrations, including roentgenograms, have been included.

The author desires to acknowledge indebtedness to his son, Dr. Arthur M. Fishberg, for collecting materials, and revising the proof of this edition.

M. F.

FROM THE PREFACE TO THE SECOND EDITION.

It is the purpose of this book to supply the general practitioner with information concerning the etiology, diagnosis, prognosis and treatment of pulmonary tuberculosis, its clinical forms and common complications. An experience of over twenty years with the tuberculosis problems in New York City has convinced the author that: (1) The physician can, and should, do more than recognize phthisis in its earliest or pretuberculous stage and at once consign the patient to a sanatorium. (2) That "incipient" does not always mean *curable* tuberculosis, and conversely, that "advanced" disease does not necessarily indicate a hopeless outlook. (3) That institutional treatment is not the only effective method of handling the phthisical patient. (4) If all tuberculous persons in this country would consent to hospitalization, the available institutions would hardly accommodate ten per cent of eligible patients. (5) Even those treated in sanatoriums must be cared for by their family physicians before admission and after discharge. (6) Careful home treatment is productive of practically the same immediate and ultimate results as institutional treatment, and is less costly to the patient and to the community.

Recent investigations of tuberculous infection have radically changed our views on the transmissibility of tuberculosis. On the one hand, it was found that patients who indiscriminately expectorate tubercle bacilli are a greater menace than has hitherto been suspected. Infants may be infected by mere contact with phthisical persons. On the other hand, there is hardly a person living in a large city who has escaped infection with tubercle bacilli. In other words, despite the vigorous and costly efforts which have been made during the past thirty years, the majority of the population in civilized countries harbor tubercle bacilli in their bodies. But, what is of more importance, not every one infected with tubercle bacilli is destined to become sick. For this reason, a sharp distinction is made in the

following chapters between *infection* and *disease*, or *tuberculosis* and *phthisis*.

Recent research has also shown that infection with tubercle bacilli endows an organism with a certain degree of resistance, or even immunity, against further and renewed exogenic infection with the same virus. Experimental investigations have proved that it is impossible to reinfect a tuberculous animal with tubercle bacilli. Many clinical phenomena, which have hitherto baffled those who studied the disease, such as the rarity of conjugal phthisis, or of tuberculous disease in those living and working among phthisical patients, and of soldiers in the armies, are now explained by this immunity of the tuberculous against reinfection with tubercle bacilli. Phthisis is at present considered a manifestation of immunity. Prophylaxis of *infection* has been shifted to the child, while that of phthisis involves more than prevention of infection.

In the discussion of the clinical aspects of phthisis an attempt has been made to elaborate on the constitutional symptoms, which are still the sheet-anchor of the physician who is charged with deciding whether a patient is ill and in need of treatment. Bacteriology and serology are excellent helps in showing whether the patient has been infected with tubercle bacilli; skiagraphy reveals airless areas of lung tissue; but they do not give conclusive proof that the patient is sick and in need of prolonged and costly treatment. We also know that unity of causation is not always an indication of unity of resulting clinical phenomena in tuberculosis: The clinical picture of tuberculosis in infants is different from that in children; in adults some, irrespective of the treatment applied, show a marked tendency to sclerosis or fibrosis of the lesion; in others caseation and destruction of lung tissue go on progressively; in still others, there is a sluggish course, marked by periods of illness alternating with periods of comparative comfort. For these reasons several types of the disease, or syndromes, have been described, each of which has not only a different clinical course but also a different outlook as to recovery, and the treatment differs markedly in each form of the disease.

It appears that the tuberculosis problem has been handled in the various armies engaged in the recent World War along the lines mentioned above and some have anticipated that the disease will prove as great a menace as many other war plagues, such as typhoid, influenza, dysentery, etc. However, despite the fact that only clinical tuberculosis has been considered cause for rejection by draft boards,

and tuberculin (the test for infection) has not been applied for diagnostic purposes at all, the number of cases of active tuberculosis in the armies has not been excessive, considering that soldiers are of the age period when the disease is most likely to occur. This clearly has been an experiment on a large scale showing that tuberculous infection is not acquired by adults; that infection, which is in the vast majority of cases acquired during childhood, is not invariably followed by disease, and that only constitutional symptoms decide whether a patient is sick with phthisis and in need of treatment. Our rather unconventional views on the diagnosis and prophylaxis of phthisis as a disease, which have been emphasized in the first edition of this book, have thus been fully confirmed. Though infection as a factor in phthisiogenesis has been practically disregarded in the various armies engaged in the recent war, no visible harm has resulted.

The treatment recommended in this book is based on experience with patients in New York City. Some were living in the congested neighborhoods of the Metropolis; others in the better parts of the city; still others have been under the author's care in the hospital. A large proportion had been in sanatoriums, but even they had to be cared for in their homes before admission and after discharge. Emphasis is laid on the fact that in most cases we can give the patient the benefit of modern and approved treatment in his home as well as in institutions. The immense utility of sanatorium treatment is emphasized and its limitations are enumerated. It is also shown that institutional treatment is not the only, nor the best, available method of caring for the majority of patients. Experience has taught that we can properly house and feed a patient in the city at a much less expense than in a sanatorium.

Medicinal treatment has been alloted some space for the reason that it is, in many cases, believed to possess more value than it has been accredited by therapeutic nihilists. The most recent method of treatment, artificial pneumothorax, has been given at some detail because of its efficacy in selected patients in whom everything else has failed to afford relief.

M. F.

CONTENTS.

CHAPTER I.

THE TUBERCLE BACILLI 17–39

CHAPTER II.

TUBERCULOUS INFECTION 40–60

CHAPTER III.

THE EPIDEMIOLOGY OF TUBERCULOSIS 61–100

CHAPTER IV.

PHTHISIOGENESIS I. PREDISPOSITION, ENDOGENOUS AND EXOGENOUS . 101–137

CHAPTER V.

PHTHISIOGENESIS II. THE PHENOMENA OF IMMUNITY 138–159

CHAPTER VI.

PATHOLOGY AND MORBID ANATOMY 160–186

CHAPTER VII.

SYMPTOMATOLOGY OF PHTHISIS—HISTORY OF THE PATIENT 187–194

CHAPTER VIII.

COUGH AND EXPECTORATION 195–212

CHAPTER IX.

FEVER AND NIGHTSWEATS 213–233

CHAPTER X.

HEMOPTYSIS 234–256

CHAPTER XI.

SYMPTOMS CAUSED BY DISTURBANCES IN THE GASTRO-INTESTINAL TRACT—THE SKIN—THE JOINTS 257–276

CHAPTER XII.

SYMPTOMS REFERABLE TO THE CARDIOVASCULAR AND RENAL SYSTEMS . 277–288

CHAPTER XIII.

NERVOUS SYMPTOMS OF PHTHISIS 289–298

CHAPTER XIV.

INSPECTION AND PALPATION 299–313

CHAPTER XV.

PERCUSSION OF THE CHEST IN PHTHISIS 314–335

CHAPTER XVI.

AUSCULTATION OF THE CHEST IN PHTHISIS 336–353

CHAPTER XVII.

ROENTGENOGRAPHY IN THE DIAGNOSIS OF PHTHISIS 354–366

CHAPTER XVIII.

THE CLINICAL FORMS OF PHTHISIS 367–373

CHAPTER XIX.

CHRONIC PHTHISIS. INCIPIENT STAGE 374–395

CHAPTER XX.

CHRONIC PHTHISIS. ADVANCED STAGE 396–413

CHAPTER XXI.

ABORTIVE TUBERCULOSIS 414–418

CHAPTER XXII.

FIBROID PHTHISIS 419–428

CHAPTER XXIII.

ACUTE FORMS OF TUBERCULOSIS 429–446

CHAPTER XXIV.

PULMONARY TUBERCULOSIS IN CHILDREN 447–473

CHAPTER XXV.

PHTHISIS IN THE AGED 474–477

CHAPTER XXVI.

TUBERCULOSIS OF THE PLEURA 478–520

CHAPTER XXVII.

PNEUMOTHORAX 521–533

CHAPTER XXVIII.

DIFFERENTIAL DIAGNOSIS OF PULMONARY TUBERCULOSIS 534–557

CHAPTER XXIX.

COMPLICATIONS OF PHTHISIS 558–574

CHAPTER XXX.

RECIPROCAL RELATIONS BETWEEN PULMONARY TUBERCULOSIS AND CERTAIN PHYSIOLOGICAL AND PATHOLOGICAL PROCESSES . . . 575–617

CHAPTER XXXI.

PROGNOSIS IN PULMONARY TUBERCULOSIS 618–633

CHAPTER XXXII.

THE MEDICO-LEGAL AND INSURANCE ASPECTS OF TUBERCULOSIS . . 634–651

CHAPTER XXXIII.

THE INDICATIONS FOR TREATMENT OF PHTHISIS 652–659

CHAPTER XXXIV.

PROPHYLAXIS 660–676

CHAPTER XXXV.

GENERAL MANAGEMENT OF THE CASE 677–686

CHAPTER XXXVI.

THE REST CURE 686–695

CHAPTER XXXVII.

OPEN-AIR TREATMENT 696–709

CHAPTER XXXVIII.

CLIMATIC TREATMENT 710–722

CHAPTER XXXIX.

INSTITUTIONAL TREATMENT 723–732

CHAPTER XL.

DIETETIC TREATMENT 733–745

CHAPTER XLI.

MEDICINAL TREATMENT 746–760

CHAPTER XLII.

SPECIFIC TREATMENT 761–770

CHAPTER XLIII.

SYMPTOMATIC TREATMENT 771–793

CHAPTER XLIV.

OPERATIVE TREATMENT—ARTIFICIAL PNEUMOTHORAX 794–843

CHAPTER XLV.

GENERAL TREATMENT OF THE VARIOUS FORMS OF PULMONARY TUBERCULOSIS 844–855

CHAPTER XLVI.

TREATMENT OF COMPLICATIONS 856–864

INDEX OF AUTHORS 865–874

INDEX OF SUBJECTS 875–891

PULMONARY TUBERCULOSIS.

CHAPTER I.

THE TUBERCLE BACILLI.

MANY ancient and medieval physicians considered pulmonary tuberculosis a contagious disease. In 1865 J. A. Villemin[1] demonstrated experimentally that the disease can be transmitted from one animal to another, and from human beings to animals by inoculation of the sputum brought up by tuberculous patients. It remained for Robert Koch[2] to isolate the microörganism which is the infective agent. In 1882 he published his first communication describing the morphology, staining reactions, cultivation, and the successful animal inoculation of pure cultures of the bacilli invariably found in tissues affected with tuberculosis.

The tubercle bacillus is a parasite in the full sense of the word, living and thriving only in the bodies of animals and man, and perishing outside of the animal body. It has not been decided to which group of microörganisms it belongs; in fact, we do not as yet have a classification of bacteria which is completely satisfactory to all who are competent to judge. It may be said to belong to the group of acid-fast bacteria, of which there are many varieties to be mentioned farther on, and may be classified with the trichomycetes, while some consider it intermediary between the true bacteria and the lower fungi, hyphomycetes.

Morphology.—The morphological variations of the tubercle bacilli are dependent on their type and virulence, whether human, bovine, or avian, and on the media in which they have been cultivated. In film preparations made from cultures, or from sputum expectorated by tuberculous patients, the tubercle bacillus appears as a slender rod, usually straight, but very often curved, about one-fourth to one-half the diameter of a red blood corpuscle, or $\frac{1}{500}$ mm. in length, on

[1] Cause et nature de la tuberculose, Gaz. hebd., 2d ser., 1865, p. 50; Bull. de l'Acad. de Méd. de Paris, 1865, **31**, 211; Études sur la tuberculose, preuves rationelles, experimentelles de sa specificité et de son inoculabilité, Paris, 1868.

[2] Berl. klin. Wchnschr., 1882, **39**. An English translation of the complete report, originally appearing in Mitt. aus dem Gesundheitsamte, 1887, vol. **2**, made by Stanley Boyd, has appeared in "Recent Essays on Bacteria in Relation to Disease," New Sydenham Society, 1886, pp. 65–201.

the average. These rods, mostly rounded on the two ends, are seen in the preparations from secretions or tissues, singly, in pairs, or in heaps, occasionally imbedded in the tissue cells. They are non-motile, and have no flagella. Microscopically, an enveloping or capsular, fatty or waxy substance can often be made out around each bacillus, especially in those which have been artificially cultivated in serum for several generations. Some individual bacilli are strikingly pleomorphic, thread, or club-shaped, with thickenings at either or both ends, or with filaments passing out from the main rod at right angles, and finally in Y-shaped branchings. But these are of no practical significance, because they appear to be simply degenerated types of the microörganism, although some look at them as the reverse, the result of active growth on a good culture medium, and amid favorable biological surroundings. In some individual bacilli, unstained spaces, regarded as vacuoles, are seen, giving the rod the appearance of a chain of cocci. Bulgings in various places, showing deeper stain, more resistant to decolorization, have been considered as spores by some authors. But this appears to be erroneous, because they have no stronger resistance than the body of the bacillus, and succumb to heat and chemicals as fast as the entire rod. The fact that it is speedily killed by sunlight also indicates that the tubercle bacillus has no spores.

Staining.—It has already been noted that the tubercle bacillus is surrounded by a fatty or waxy capsule. It is to this sheath that the peculiar staining reactions of the bacillus are attributed.

The tubercle bacillus shows but little affinity for ordinary stains, not combining with them unless exposed for a long time, or heated. Koch originally stained the bacilli in sections with alkaline methylene blue, counterstaining with Bismarck brown. But the first practically useful method was that of Ehrlich who found that if the bacilli were stained with aniline water and fuchsin, or gentian violet, they subsequently resisted decolorization with nitric acid and alcohol. Hence the designation of the bacilli as *acid-fast.* They were subsequently also shown to be alkali fast. The stains in use at present all rest on this principle of intense staining followed by decolorization vigorous enough to remove the stain from everything on the film but the

EXPLANATION OF PLATE I.—(Park and Williams.)

FIG. 1.—Tuberculous lymph node "giant cell" containing tubercle bacilli "human type." Bacilli red, rest of specimen blue. Ziehl-Neelsen stain. × 1000 diam.

FIG. 2.—Tuberculous sputum from human case. Stain same as above. × 1000 diam.

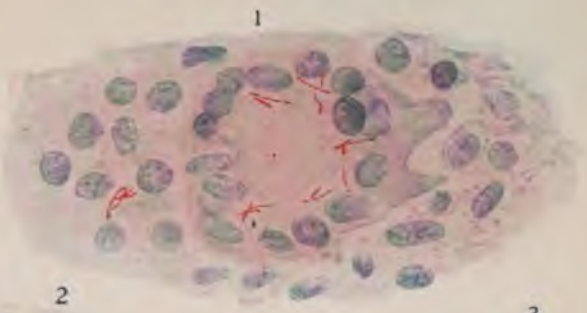
FIG. 3.—Tuberculous sputum, human case. Stained by Hermann's method. Tubercle bacilli violet, rest of specimen brown.

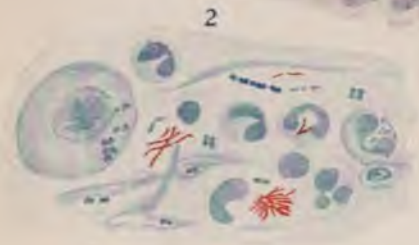
FIG. 4.—Pus from tuberculous abscess in cow. "Bovine type" of bacillus. Stained same as Figs. 1 and 2. × 1000 diam.

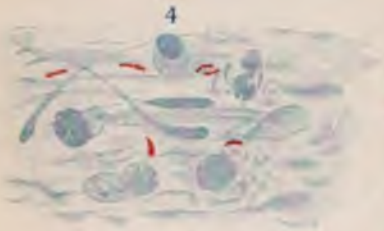
FIG. 5.—Section through leprous skin showing bacilli in clumps in and out of cells and large "leprous cell" containing a ball of bacilli. Stained with Ziehl-Neelsen.

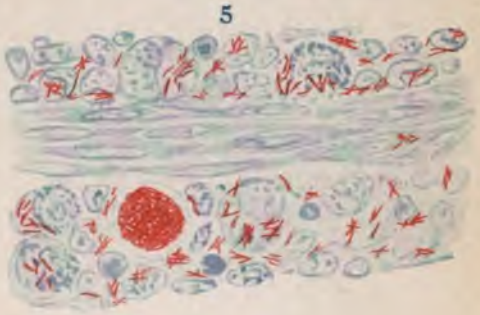
FIG. 6.—Photograph of human type of tubercle bacilli from sputum. Bacilli in red, rest of specimen blue. × 1000 diam. (Fränkel and Pfeiffer.)

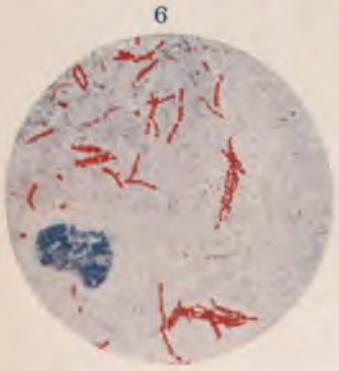
PLATE I











tubercle bacilli, whose lipoidal sheath tenaciously holds the coloring matter.

The *Ziehl-Neelsen method* is the most popular. The stain used is Ziehl's carbol-fuchsin solution, prepared as follows:

Fuchsin	1 gram
Absolute alcohol	25 c c
Carbolic acid, 5 per cent solution	100 c c

1. Make a rather thin smear on a slide or, preferably, on a cover slip. Fix by heat. Flood the staining solution, and steam (not boil) over a flame for about three minutes, replacing evaporated solution.
2. Decolorize with 2 per cent hydrochloric acid, or 10 per cent sulphuric acid solution, until the red of the thinner portions has altogether disappeared, and the preparation in general appears pink.
3. Complete the decolorization by washing thoroughly in 95 per cent alcohol; then wash in water.
4. Counterstain with a 1 per cent aqueous solution of methylene blue; wash, dry, and examine with an oil-immersion lens.

Gabbet has modified the Ziehl-Neelsen stain by combining the decolorization and counterstaining. He stains as above with Ziehl's carbol-fuchsin, and after allowing the excess of stain to drain off, the following solution is applied for two or three minutes:

Methylene blue	2 grams
Concentrated sulphuric acid	10 cc
Water	100 cc

Wash in water and dry.

With both the above methods the tubercle bacilli appear bright red, all else in the field being blue (see Plate I). Gabbet's method, while simpler than that of Ziehl and Neelsen, is not so reliable. For diagnostic purposes the Ziehl-Neelsen method remains the most useful.

The tubercle bacillus is Gram-positive, but this method of staining does not find wide practical application.

Clinically, it is at times necessary to find tubercle bacilli in specimens in which they are very scanty, and the above methods fail to reveal them though they are present. In such cases concentration and sedimentation of the specimen is practised. For these purposes the antiformin method has recently found wide application. The details of this procedure are discussed later on (see p. 204).

It must be mentioned that the tubercle bacilli are not the only known acid-fast bacilli. This is one of the sources of error which, at times, interferes with the proper appreciation of acid-fast microörganisms discovered under the microscope.

Much's Granules.—For a long time it has been noted that in the lesions in tuberculous cattle, be they as characteristic macroscopically as possible, no tubercle bacilli can be found microscopically, but inoculation experiments prove positive almost invariably, thus showing

that virulent germs must have been there. Likewise, in human pathology, it has often been observed that in cold abscesses no tubercle bacilli can be found, and again inoculation of the purulent material infects animals. These and other facts have led H. Much to suspect that there are bacilli which produce specific tuberculous lesions though the germs are not acid-fast. Research has, in fact, convinced him that he found tubercle bacilli which, while remaining virulent, have lost their acid-fast characters. Hans Much,[1] by whose name these microörganisms are generally known, describes two forms of these granules: (1) A rod-shaped granular organism; (2) isolated granules; both of which cannot be stained by the Ziehl method, but only by the Gram method. They are pathogenic to animals and man, and are usually found in some cases of slowly progressing chronic phthisis, fibroid phthisis, cold abscess, etc. It is thus evident that before concluding that a given case lacks acid-fast bacilli, and is therefore not tuberculous, the Much granules are to be looked for by staining with the Gram-Much method. According to William H. Park, true tubercle bacilli are probably always present together with the granules in cases in which the latter forms are found.

In this country Charles N. Meader[2] has made a careful study of these granules. In his opinion "the biological relationship of Much's forms of tubercle bacilli is a matter of considerable interest. They may be considered as a natural stage in the evolution of the bacillus, as the result of degenerative changes, or may be classed as spores (*i. e.*, as resisting forms). The accumulated evidence tends to show that they are found predominant in tissues of a distinctly fibroid character, in old cavities, in pus of cold abscesses, in old cultures, in the notably indolent lupus lesions and in sclerosed lymph glands—facts which, taken together, mark them as forms assumed under unfavorable conditions, whether they be the result of sporulation or of degeneration. The same conclusion is suggested by observations that, under favorable cultural conditions, they are rapidly replaced by Ziehl-staining forms. Against their classification as spores, in the commonly accepted sense, is the fact that the granular forms are rather less resistant to the action of antiformin than are the Ziehl forms; their resistance to other chemical agents has not yet been reported upon. It is of interest to note here that the granular forms appear more frequently in the bovine than in the human type of bacillus."

Cultivation.—The tubercle bacilli are obligatory aërobes; they require free oxygen for maintenance of life, activity and propagation. In artificial media they grow very slowly, much more slowly than most bacteria which are not acid-alcohol-fast; they proliferate very slowly, and other saprophytic microörganisms which happen to live with them soon outnumber them. Working for the British Royal Commission, A. S. Griffith succeeded in perfecting a method which avoids contami-

[1] In Brauer, Schröder, and Blumenfeld's Handbuch d. Tuberkulose, **1**, 193.
[2] Am. Jour. Med. Sci., 1915, **101**, 858.

nation, by the use of antiformin. He inoculates successive cultures of tuberculous material, mainly sputum, and antiformin which inhibits, or even prevents, the growth of extraneous microörganisms. In this country Petroff's medium is used to facilitate the isolation of tubercle bacilli from sputum, feces, and other contaminated sources. This selective medium is based on the fact that the lipoid capsule of the tubercle bacillus enables it to resist the action of alkalies much better than bacteria which lack this means of defense. He also takes advantage of the selective inhibitory action of certain dyes on the growth of bacteria.

Once the difficulties have been overcome and the bacilli have been cultivated, it is rather easy to transplant them to another culture of the same medium, and growth is even more luxuriant in the subsequent cultures. Theobald Smith's method of cultivation on dog serum and Dorset's egg medium, and especially Petroff's medium, are about the best and most used in this country. Pure cultures are best obtained from tubercles of animals inoculated with the bacilli. But it is often possible to obtain pure cultures from closed tuberculous cavities, from lesions of lupus, and even from sputum.

When cultivated on coagulated dog serum, or bovine serum, or in Dorset's egg medium, especially when to the latter there is added glycerin, growth appears usually at the end of ten days at 37° C., and within four weeks the characteristic growth may be expected. On the glycerin-egg medium the human form of organism produces an abundant, wrinkled layer, usually having a yellowish, buff, or pinkish color. The growths are seen as more or less elevated colonies which may coalesce. On glycerin-agar the growth is more rapid than on serum, and appears as a thick, white layer, becoming yellowish. Tubercle bacilli also flourish in glycerin-potato medium.

Powers of Resistance.—The tubercle bacilli grow best at the temperature of the human body, 37° to 38° C., but growth is not abolished at 29° to 42° C. From a practical standpoint it is important to mention that they are not killed when exposed to moist heat of 50° C. for less than twelve hours, but heating to 55° C. for four to six hours does destroy them. They are also killed when exposed to moist heat of 60° C. for one-half hour, and in fifteen minutes at 70° C.; in five minutes at 80° to 90° C., and in one minute at 95° C. With sputum, conditions are different: the mucus protects the bacilli and it requires more time to destroy them with heat. However, five minutes' boiling is sufficient to kill the bacilli under all circumstances.

Another practical point is that in milk, tubercle bacilli resist the action of heat with greater tenacity than in pure liquid cultures, or even in sputum. From many careful experiments it appears that heating milk for thirty to forty minutes, at a temperature of 65° to 70° C., or boiling for three minutes, destroys tubercle bacilli. Especially resistant are the bacilli when the milk is heated in an open vessel and a pellicle forms on the top of the fluid. This protects the bacilli against

a temperature of 60° C. for an hour. William H. Park explains this by the fact that the upper parts of the fluid are not heated to the same degree as the lower, and some bacilli may survive. At any rate, it is important that pasteurization should be done in closed vessels. In butter the virulence of the bacilli is greatly diminished and even abolished when in contact for a long time. In fact, they die out within a few weeks, as a rule. The reasons for this phenomenon are not clear. On the other hand, Schroeder and Cotton have found living tubercle bacilli retaining their virulence for one hundred and sixty days in salted butter when kept without ice in a house cellar; and Mohler, Washburn, and Doane found that they survived a year in cheese. In thoroughly boiled or roasted meat the bacilli are destroyed; but in the rare portions they may survive. Sausages, etc., made of uncooked meat, may contain living tubercle bacilli.

Dry heat is less potent in destroying tubercle bacilli; circulating steam requires one-half hour for this purpose, while bacilli in dried sputum can withstand a temperature of 100° C. for an hour. On the other hand, cold does not destroy their virulence, and freezing, with subsequent thawing, does not harm them very much.

As has already been mentioned, the fatty substances and wax contained in the tubercle bacilli protect them to a certain degree from the effects of desiccation, and from the bactericidal action of the normal body cells, although for growth and proliferation they require moisture. When dried and pulverized by being converted into dust, as is often the case with tuberculous sputum eliminated indiscriminately by careless patients, most of the bacilli succumb, but some have been found to resist desiccation at ordinary temperature for months.

In this connection it must be borne in mind that the action of light is an important factor. It has been ascertained that light, especially sunlight, decomposes the fatty substances in the bacilli and thus destroys them altogether. When cultures are exposed to direct sunlight for a couple of hours, the vitality as well as the virulence of the tubercle bacilli is destroyed; in sputum the bacilli are protected by the mucus, and it requires a longer time for their destruction. Some maintain that their virulence is destroyed with only partial loss of vitality.

On the other hand, recent investigations by Tècon[1] show that tubercle bacilli expectorated in sputum remained virulent for as long as nine days though exposed to the action of the sun in summer, and he concludes that the sterilizing action of sun rays has been overestimated; in the clumps of sputum they may survive for days.

These facts have been utilized in attempts at prophylaxis of tuberculosis. The danger of expectoration of tuberculous sputum is less in streets and in well ventilated rooms where bright light, especially sunshine, sooner or later renders it harmless, than in dark rooms

[1] Paris Médicale, 1920, 10, 33.

where the bacilli may retain their vitality and virulence for as long as one year, and even drying does not harm them very much.

On the whole, tubercle bacilli may retain their vitality for a considerable time if not in exceptionally unfavorable surroundings. In the latter case their growth is soon hampered, and they cannot successfully be transferered by inoculation to another culture medium; but they may retain their virulence much longer and cause disease when inoculated into animals. After several months, however, even this wanes, and after six months this property is completely lost. Raw[1] demonstrated that pure cultures of human, bovine, and avian bacilli may retain all their characteristics and selective appearances for as long as twelve years when regular subculturing on artificial media containing glycerin is practised. In laboratories it has been found by experience that it is safer to reinoculate cultures every four to six weeks. Exceptionally, cultures have been found alive and virulent after two years. This is especially the case with potato and bouillon cultures which have been kept under favorable conditions, as to heat, moisture, etc., while in serum and glycerin cultures the bacilli do not survive so long.

Cornet found that serum cultures remain alive for about six months, while glycerin-agar cultures are often partially, or wholly, dead in six to eight weeks. There seems also to be some difference in this respect between the various types of tubercle bacilli: Maffucci states that avian bacilli may remain alive for two years, and Strauss found that cultures of human tubercle bacilli are only exceptionally capable of reproduction after five to six months; after eight to twelve months they fail regularly. Theobald Smith[2] found that a culture three months old failed, as a rule, to yield successful subcultures, and that tubercle bacilli, of both human and bovine types, when kept in fully developed cultures at 40° to 50° F., may remain infectious to guinea-pigs for from seven to nineteen months, but the number of bacilli surviving in such cultures is relatively small. Delépine's[3] experience has been that tubercle bacilli retain some of their pathogenicity as long as 500 days if left in the dark in milk at a low temperature, below 6° C.; but after being kept thus for four and one-half years these bacilli were no longer pathogenic to guinea-pigs. It is, however, important to bear in mind, when considering prophylaxis, that when tubercle bacilli in sputum are deposited in dark rooms they may retain their vitality and power to cause disease for as long as three hundred and nine days, as has been found by Soparkar.[4] The oldest tuberculous sputum which has been investigated was that reported by Newell Bly Burns.[5] He examined sputum twenty-two years old and found that the bacilli re-

[1] Lancet, 1919, **1**, 376.
[2] Jour. Med. Research, 1913, **28**, 91.
[3] Ann. de l'Inst. Pasteur, Paris, 1916, **30**, 600.
[4] Indian Jour. of Med. Research, 1916, **4**, 62.
[5] Amer. Review of Tuberc., 1917, **1**, 484.

tained their staining qualities, but lost completely their power to grow and their pathogenicity.

Tubercle bacilli display great powers of resistance to the action of the products of other bacterial growths, in spite of the fact that they have no spores. They may survive for months in souring milk, in sewage and in water, and in putrefying matter generally, especially sputum. Lawrason Brown, S. A. Petroff and F. H. Heise[1] found virulent tubercle bacilli in the water of the Saranac River, into which the sewerage system of Saranac Lake empties. Every sample of water taken from below the surface, from the outlet of the sewer to a point three and a half miles down the stream, showed the presence of acid-fast organisms. No acid-fast organisms were found above the outlet or twelve miles below the outlet. The bacilli were believed to be derived from the feces of the numerous tuberculous individuals in Saranac. In fact, where no particles of feces were discovered in the water, no viable tubercle bacilli were found.

Virulence.—Long before the discovery of the tubercle bacillus it was known that certain diseases in animals were of the same character as human tuberculosis, and attributed to the same virus. Klenke, in 1846, emphasized the danger of milk from tuberculous cattle as an infective agent to human beings, and Villemin, in 1865, showed by animal experiment that tuberculous disease in man and animals is identical in character. With the study of the virulence of the tubercle bacillus it was found that it is pathogenic to many species of animals. In some, tuberculosis is known to occur spontaneously, while others may be infected artificially. There appear to be significant differences in the results of such experimental infections, depending on the method of inoculation of the virus—injections into the subcutaneous tissues, into the peritoneum, into the anterior chamber of the eye, intravenously, by feeding animals with bacilli, or compelling them to inhale the bacilli with inspired air, and also according to the origin of the bacilli. However, virulence is hardly, if at all, influenced by passage through animals. Baldwin, Krause, and others worked with a culture for over ten years and they never noted any variation of its virulence.

Tubercle bacilli obtained from different cases of human tuberculosis often show differences in their virulence according to the strain. Some authors have observed differences in virulence according to the origin of the bacilli from various tuberculous diseases, and they have then attempted to attribute the numerous clinical phenomena of this disease to the degrees in virulence of the virus. But, so far, no conclusive proof for such assumptions have been brought forward. Inoculating numerous animals with sputum derived from one tuberculous patient, there are often observed different results, showing that the susceptibility of the individual animal must be reckoned with. More-

[1] Tr. Nat. Assn. Prevent. Tuberc., 1916, **12**, 286.

over, we should not apply, without careful consideration, results of animal experimentation to clinical phenomena in human beings.

When the bacilli obtained from different animals are compared, the difference in their virulence are even more striking. For this reason there have been described different species, varieties or strains of tubercle bacilli, although some authors maintain that the differences in cultural and virulence characteristics are acquired while the microorganisms are sojourning in the host by adaptation to the conditions favorable for their growth.

HUMAN, BOVINE, AND AVIAN BACILLI.

In the early history of the scientific investigation of tuberculous infection it was already noted that there are some differences between human and bovine tubercle. Villemin was the first to mention these differences. "We must note that none of our rabbits," he said, "inoculated with human tubercle have presented a tuberculization so rapid and generalized as that which we have obtained with material from the cow. At first we were inclined to regard this as fortuitous, but subsequent experiment led us to suppose that the tubercle of the bovine race inoculated into rabbits possesses a much greater activity than that obtained from man. It may be supposed that, like all virulent matter, the tuberculous matter is the more virulent the more the affinity of the animal supplying the virus and the animal receiving it." This apparently was entirely forgotten, until Nocard and Roux, and Rivolta and Maffucci again rediscovered it while doing inoculation experiments with tubercle bacilli derived from humans and from cattle.

It remained, however, for Theobald Smith[1] to make the first careful study of differences in morphological, cultural, and pathogenic types of tubercle bacilli. In 1898 he showed that there are differences between the bacilli isolated from human beings, when compared with those isolated from cattle. His designation of the former as "human," and the latter as "bovine," has since been generally accepted. In 1901 Robert Koch also announced that his studies led him to the conviction that human and bovine tuberculosis are not identical; that the bovine bacilli are, in fact, not pathogenic to man, and that no special measures need be taken to protect man against the consumption of milk and meat from tuberculous cattle. Considering the commercial interest which is centered around this problem, in addition to the problem of human infection, it is clear why studies along these lines have been in abundance during recent years.

Still other types of bacilli have been found. Rivolta and Maffucci have shown that there are certain morphological and biological differences between the tubercle bacilli found in birds and those in human beings. Theobald Smith continued the investigation of the

[1] Jour. Exper. Med., 1898, **3**, 451.

problem and arrived at the conclusion that bacilli from human sources are not clearly identical in every respect with those obtained from bovine sources. Official bodies of the Imperial Department of Health in Germany, a Royal Commission in England, and Dr. William H. Park, for the New York City Department of Health, have thoroughly studied the problem, each from a different angle. The result is that we are at present in a position to state conclusively that there is more than one variety of tubercle bacillus.

The conclusions of the British Royal Commission are to the effect that "for the purpose of description it is advantageous to distinguish three types of tubercle bacilli, recognizable by their individual characters. These are the human, the bovine, and the avian. The human type, although so named, is not the only one found in cases of tuberculosis in man. It is the organism present in the majority of such cases, but in some cases of human disease the bacilli present are of the bovine type, and in others the bacilli have special characters distinguishing them from each of the three principal types. In natural cases of tuberculosis in cattle the only type of bacillus present is the bovine type." William H. Park[1] concludes from his extensive study of the subject that "tubercle bacilli, as isolated from man, fall into two groups. One of these groups is identical in all its characters with those found in cattle. That is, all tubercle bacilli from man and cattle fall into two groups, which have been designated the human and bovine types."

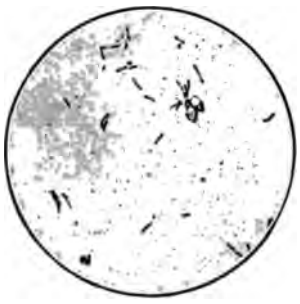


FIG. 1.—Tubercle bacilli, human. × 1000 diameters. (Park and Williams.)

Human Bacilli.—The human variety grows on all culture media quickly and luxuriantly; the addition of glycerin enhances its growth. On glycerin bouillon growth is seen during the first few days, and within three weeks there is seen a pellicle on the surface of the culture which spreads laterally and reaches the glass walls. The pellicle is fragile and its surface wrinkled. Morphologically, the human bacilli, when grown on serum cultures, appear as long, straight or curved rods which are unevenly stained.

[1] Jour. Med. Research, 1911, **20**, 313; 1912, **22**, 109.

In general it may be stated that the virulence of human bacilli is rather low in various animals. Guinea-pigs are very susceptible and may be infected in various ways, even by rubbing the bacilli into the shaved skin of the abdomen. Rabbits are, however, less susceptible. Even when a milligram of bacilli is injected into a vein of the ear there is only produced a chronic lesion which may heal; subcutaneous injection produces an infiltration at the point inoculated, which soon softens and empties itself through a fistulous opening, or may even be absorbed. The regional lymph glands swell, but do not caseate. At times, but not in every case, there may thus be produced a chronic infection of the lungs in the rabbit. Intraperitoneal inoculation produces tuberculous peritonitis, which may extend along the diaphragm; infection of the anterior chamber of the eye produces a lesion which develops more slowly than when bovine bacilli are used. Cattle are infected when large doses are injected intravenously. But with subcutaneous infection there is produced only an infiltration at the point inoculated, which soon suppurates and heals. The regional lymph glands swell up and at times become calcified. Feeding calves with human bacilli never produces any progressive disease. Pigs, dogs, cats, and sheep are not at all affected by human bacilli, while monkeys are very susceptible. Some species of birds are also susceptible.

FIG. 2.—Tubercle bacilli, bovine. × 1000 diameters. (Park and Williams)

Bovine Bacilli.—The bovine bacilli are very difficult to cultivate; it appears that the addition of glycerin to the culture medium slackens their growth. On glycerin bouillon growth is very slow. A thin pellicle is formed which spreads all over the surface within four to eight weeks, but it may remain limited to the center of the surface. Only rarely are a few verrucose thickenings formed on the surface. After several transplantations they may show greater tendencies to grow. Morphologically, they appear as shorter, thicker, are more evenly stained than the human variety, and usually bent, showing beading and irregularities in staining. Park, who has done excellent work along these lines, says: "Although one could in many instances make a probable diagnosis of type from an inspection of the smear, the

number of intermediate gradations in morphological differences rob it of nearly all its practical value."

The bovine bacilli are more virulent for rabbits, calves, and swine than the human. Guinea-pigs are just as susceptible to them as they are to the human variety, but in addition they are killed, or become acutely and progressively sick, when infected with small doses of bovine bacilli. The difference in the virulence of the two types is well seen in the rabbit. The bovine type of virus causes in every instance generalized miliary tuberculosis, progressive, terminating in death of the animal. "Human virus injected in the same amount produces either no disease at all, or lesions of varying severity in the lungs or kidneys, or both, and never causes generalized miliary tuberculosis. Even with 1 mg., that is, one hundred times as much, the lesions are usually confined to the same organs, and though there is a very slight tendency to generalization with this dose, there is never a generalization showing a progressive nature. Rabbits injected even with the larger dose live indefinitely, and, if death should occur, the tuberculous lesions are usually not extensive enough to say that the animal died of the disease." (Park and Krumwiede.)

Cattle are very susceptible to the bovine virus, and after intravenous injection perish from generalized tuberculosis within three or four weeks. Intraperitoneal, intraocular, and intramammary inoculation also cause generalized and fatal tuberculosis. Feeding cattle with even small doses of pure culture of bovine tubercle bacilli causes tuberculous disease of the intestines, followed by tuberculous lymphangitis and lymphadenitis of the mesentery; the disease spreads to other lymph glands, serous membranes, and lungs. Inhalation produces caseous pneumonia. After subcutaneous injections there is produced an infiltration at the point inoculated, swelling of the regional lymph glands, and generalized tuberculosis, the animal perishing within two or three months. Pigs, sheep, goats, cats, and monkeys are very susceptible; dogs, rats, and mice are more or less refractory. Some species of birds are susceptible, but chickens show complete resistance.

Avian Bacilli.—On glycerin agar and on serum their growth is more luxuriant, appears more moist, or slimy, than observed in mammalian bacilli, and they produce an orange pigment. They grow at the temperature of 41° C., which stops the growth of mammalian tubercle bacilli. Morphologically, the differences are insignificant. The Royal Commission found that rabbits, rats, and mice are the only mammals susceptible to inoculation with avian tubercle bacilli. Fowls are very susceptible when fed with portions of the organs containing avian bacilli, but they may consume enormous quantities of phthisical sputum without becoming tuberculous. On the other hand, the parrot is susceptible to both human and bovine bacilli as well as to avian, and spontaneous tuberculosis may be due to any of the types. Tuberculosis is very common among domesticated birds and there have been observed veritable epidemics of the disease in poultry yards.

Lamallerée, Löwenstein, Kruse, Lydia Rabinowitsch, and Max Koch have reported cases of tuberculosis in human beings caused by avian tubercle bacilli.[1]

Tubercle Bacilli of Cold-blooded Animals.—Certain diseases observed in worms, lizards, frogs, turtles, snakes, and fish have great resemblance to human tuberculosis and in many cases acid-fast bacilli have been isolated. These microörganisms grow luxuriantly at the room temperature, the growth being thick and moist like that of avian bacilli, and a higher temperature than 30° C. inhibits their growth. While they do not grow at the body temperature, it appears that some have been able to acclimate them to a temperature of 36° C. Weber and Taute have cultivated this microörganism from mud, and also from healthy frogs. They therefore conclude that these acid-fast bacilli have nothing in common with tubercle bacilli, but they are saprophytes which may be found in healthy animals and in the soil. Others, however, consider them as true pathogenic bacilli of cold-blooded animals, or such as have become attenuated in their virulence by a long residence in, and adaptation to growth at a lower temperature.

Attempts have been made to use these bacilli for the purpose of immunization against infection with mammalian tubercle bacilli, but they were unsuccessful. F. F. Friedmann has even claimed that bacilli obtained from turtles are curative of existing tuberculous disease, but the results obtained have not justified in the slightest his pretensions.

Other Acid-fast Bacilli; Pseudotubercle Bacilli.—The tubercle bacilli are not the only variety of microörganisms which, once stained, refuse to be decolorized by acids and alcohol. There have been found many others presenting the same staining reactions as the tubercle bacilli, and there is no doubt that they may bring about confusion in diagnosis. Of these we may mention the following:

The most important clinically is the *smegma bacillus*, a slender, slightly curved rod, not unlike the tubercle bacillus, but distinctly shorter. It resists the action of acids and alcohol after staining. It is found in the secretions of the external genitals, mammæ, etc., especially when these secretions contain fatty matter. There have been reported cases in which extirpation of kidneys was performed as a result of mistaking these microörganisms for tubercle bacilli. It has also been described as occurring in the sputum, and in the pharyngeal and tonsillar secretions on rare occasions. It is somewhat more readily stained than is the tubercle bacillus, and considerably more easy to decolorize. For the differentiation of the smegma from the tubercle bacillus, Pappenheim devised a stain depending on the fact that the former is decolorized by prolonged exposure to rosolic acid and alcohol, while the latter is not. The smegma bacillus is probably identical with

[1] For details see, E. Löwenstein: Vorlesungen über Tuberkulose, Jena, 1920, p. 63.

the acid-fast bacillus discovered by Lustgarten in 1884, and believed by him to be the cause of syphilis.

The *Hansen bacillus*, which is the cause of leprosy, is also acid-fast. Morphologically, it is very similar to the tubercle bacillus, though a little plumper and less apt to have a beaded appearance. In staining properties it only differs quantitatively from the tubercle bacillus, not being so strongly acid-fast.

Möller's *grass bacilli* are found in infusions of timothy-grass (*phleum pratense*), resemble morphologically the tubercle bacilli, and are acid-fast. Inoculations produce lesions exquisitely resembling tubercles.

Möller has also described a bacillus found in milk, at times in pasteurized milk, according to Kuthy. Its similarity to the tubercle bacillus is more pronounced than most of the other pseudotubercle bacilli. Inoculated into the peritoneal cavity of guinea-pigs, white mice, and frogs, these pseudotubercle bacilli obtained from tonsils, tongue, and throat, produced lesions which had great similarity, microscopically, to real tubercles, but they never spread beyond these areas. The only difference which can be discovered is that while genuine tubercles are of a proliferative character, these pseudotubercles are of a more exudative and inflammatory character, showing a tendency to abscess formation.

There have been isolated microörganisms from cow's milk, butter, and from the surface of domestic animals, which morphologically, culturally, and even on inoculation, resemble tubercle bacilli. The butter bacilli, first described by Petri and Rabinowitsch, may be mistaken for tubercle bacilli even when judged by the lesions they produce on inoculation into guinea-pigs.

There have also been isolated acid-fast rods from the excrement of cattle, swine, sheep, guinea-pigs, white mice, chickens, dogs, etc. In fact, they are so frequent in the soil that any being, or thing, coming in contact with the soil is likely to have acid-fast rods, when carefully examined with the microscope. They were also found in the dust of human dwellings, in tap water, in centrifuge tubes, in the sediment of distilled water in laboratory flasks; finally, in cerumal tartar on the teeth, and in the cerumen of the human ear, and also in the mouth-pieces of musical instruments.

The acid-fast rods found in the mouth pieces of musical instruments have recently been studied by Bruno Lange,[1] who believes that they are derived from the fat used in lubricating the instruments, or the water used for washing them, or from the mouths of the musicians. These saprophytes appear to be very widely distributed. According to Lange they proliferate very slowly, and morphologically they are akin to the acid-fast bacilli derived from turtles; they are short and plump, as a rule; but long and slender forms, exactly like tubercle bacilli, are at times encountered. They grow best at a temperature

[1] Deut. med. Wchnschr., 1920, **46**, 763.

between 20° and 30° C.; some grow at 0° to 37° C. All culture media are suitable, but those containing glycerin are best.

It is interesting that inoculation of material containing acid-fast bacilli derived from musical instruments into guinea-pigs produced no general reaction, or constitutional disease, nor could it be established that these organisms proliferated within the body. Inoculation of pure cultures into guinea-pigs and white mice produced abscesses when the dose was large. After intraperitoneal injection, fibrinous and purulent peritonitis resulted which, however, healed with scar formation. Repeated inoculations caused death of the animal. At times the tissue reactions reminded of the gross appearances of real tuberculous lesions. It is noteworthy that inoculation of large doses caused swelling, not only of the regional lymph glands, but also of other glands, the axillary, bronchial and mesenteric.

It is very difficult to differentiate the pathogenic from the non-pathogenic acid-fast rods, or bacilli, microscopically; even the most experienced often cannot tell the difference when examining a smear on a slide, which shows that clinically, when the non-pathogenic bacilli are found, they are apt to lead to erroneous diagnoses. So far bacteriologists have relied on results of inoculation experiments: With the pathogenic bacilli tuberculous lesions are produced in guinea-pigs, while the others are harmless. Recently Schlossberger and Pfannenstiel[1] have shown that differentiation may be attempted by a determination of the optimal temperature for their growth, and the temperature limits of their growth. Pathogenic tubercle bacilli do not grow at a temperature higher than 42° C., while all non-pathogenic acid-fast cultures, with the exception of the "trumpet bacilli" (see p. 30), keep on growing at 50° C. They also found that pathogenic tubercle bacilli from cold-blooded animals stop growing at 37° C., thus indicating that the turtle bacilli, which Friedmann has been using for therapeutic purposes, cannot be regarded as genuine tubercle bacilli from cold-blooded animals.

It seems that the cellular structure of these pseudotubercle bacilli is closely related to that of the pathogenic microörganisms, at any rate, chemically, as is clearly shown by their similarity in staining reactions, and their effects locally when inoculated into animals. Some produce lesions not unlike those produced by the virulent tubercle bacilli, excepting that the general toxemia is lacking, and the lesion does not extend beyond the point of inoculation, though to this there are exceptions, as is the case with the trumpet bacilli. It has been stated that animals sensitized to any type of the non-virulent acid-fast bacilli are also to some degree sensitized to the virulent form. But whether they are phylogenetically related, *i. e.*, whether they have evolved from a common ancestry, has not been established. That they have not differentiated because of the variety of environment in

[1] Deut. med. Wchnschr., 1920, **46**, 1213.

which they have lived for many generations, appears to be proved by the fact that efforts at making them pathogenic by passage through the bodies of various animals for several generations have failed. However, Igersheimer and Schlossberger,[1] recently succeeded in rendering them pathogenic by successive passage, and prolonged sojourn in guinea-pigs; the lesions produced were like those of genuine tubercle bacilli. Lange,[2] working with various other acid-fast saprophytes, such as turtle bacilli, trumpet bacilli, etc., could not obtain these results.

OCCURRENCE OF THE VARIOUS TYPES OF TUBERCLE BACILLI.

The human type of tubercle bacilli is found in the vast majority of cases of all forms of tuberculosis in human beings; in adults, pulmonary phthisis is almost exclusively caused by this virus. In spontaneous tuberculosis in hogs a small percentage shows this type of bacilli, and many species of animals, especially those coming in contact with man, also are occasionally infected with human tubercle bacilli. This is the case with parrots and some animals in zoölogical gardens in cities, like lions, antelopes, gnu, chimpanzees, macacus rhesus, etc., have been found infected with the human bacilli. The dog, rat, and mouse are practically immune, while the calf, rabbit, hog, and goat occupy intermediate positions.

The bovine type of tubercle bacilli is responsible for disease in domestic animals, as cattle, sheep, goats, horses, etc. In most cases of tuberculosis in pigs, cats, and dogs, and in many cases in monkeys, the bovine bacilli are found.

The avian type is found in the vast majority of tuberculous infections in birds. Not only are fowls affected, but also birds in zoölogical gardens are susceptible and are often sick as the result of infection with this virus. Spontaneous tuberculosis in horses, swine, monkeys, cattle, mice, and rats has been found, at times, to be due to this type of bacillus. We have already mentioned that they, in extremely rare instances, have caused disease in human beings.

Bovine Type of Bacillus Tuberculosis in Man.—Of great importance is the occurrence of bovine infection in human beings. Since Koch stated the bovine bacilli were not at all identical with the human, and that they were not pathogenic to man, various investigations have been made with the result that Koch was, on the whole, not sustained. There is evidence to the effect that many cases of tuberculosis in human beings, especially in children, are due to the bovine virus. The largest collection of cases of tuberculosis of various forms was published by B. Möller,[3] comprising 2048 patients. In adults only 2.1 per cent of bovine bacilli were found, and most

[1] Deut. med. Wchnschr., 1921, **47**, 526.
[2] Ibid., p. 528.
[3] Veröff. Koch-Stiftung, 1916, Hefte 11 and 12.

of these were cases of abdominal and glandular disease, "digestive tuberculosis." In tuberculosis of the lungs only 0.51 per cent showed bovine bacilli. Of 186 cases of bovine infection, 145 were found in children under sixteen years of age, and of these, 101 had disease of the abdominal viscera, especially the cervical and abdominal glands. He also found that when bovine infection occurs in humans, it pursues a favorable and benign course. Another collection of reported cases was published by Park and Krumwiede, embracing 940 instances of tuberculosis carefully studied as to the type of organism present, and it appears that in adults, sixteen years of age and over, only tuberculosis of the skin, abdominal organs, and general tuberculosis of alimentary origin may, at times, be caused by bovine bacilli. It is, however, a fact that but comparatively few cases have been investigated, and there is a lurking suspicion that in a larger series of cases the proportion would be much smaller. On the other hand, among 778 cases of pulmonary tuberculosis only 3, or 0.4 per cent were found with bovine bacilli, showing conclusively that as regards phthisis, the bovine type of bacilli is not to be considered a vital factor in the pathogenesis of the disease.

PERCENTAGE OF INCIDENCE OF BOVINE TUBERCULOSIS IN 940 CASES, OF WHICH 7/8 WERE PULMONARY TUBERCULOSIS (PARK AND KRUMWIEDE).

Diagnosis.	Adults 16 years and over. Per cent.	Children 5 to 16 years. Per cent.	Children under 5 years. Per cent.
Pulmonary tuberculosis	0.4	0.0	2.8
Tuberculous adenitis, cervical	2.7	38.0	61.0
Abdominal tuberculosis	20.0	53.0	58.0
Generalized tuberculosis, alimentary origin	14.0	57.0	47.0
Generalized tuberculosis	0.0	16.0	8.6
Generalized tuberculosis, including meninges, alimentary origin	0.0	0.0	66.0
Tuberculous meningitis (with or without generalized lesions other than preceding)	0.0	0.0	4.6
Tuberculosis of bones and joints	3.3	6.8	0.0
Tuberculosis of skin	23.0	60.0	0.0

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Age period.	Number of cases.	Per cent of bovine bacilli.
0 to 5 years	221	37.55
5 to 10 "	313	29.45
10 to 16 "	150	14.66
16 upward "	384	6.25
Total	1068	20.70

Of the various forms of tuberculous disease in which bovine tubercle bacilli were found by Griffith the following are of interest: Bones and joints, 19.7 per cent; genito-urinary organs, 17.65 per cent; cervical glands, 46.3 per cent; meninges, 20 per cent; scrofulodermia, 34.65 per cent; lupus, 48.9 per cent.

Investigations for the Local Government Board showed that 18.4 per cent of the children under ten years who died of tuberculosis, or other causes, were infected with bovine tubercle bacilli. The predilection of the glands by the bovine bacilli is also shown in the following figures: In a series of cervical gland cases investigated by Griffith,[1] 71.4 per cent (20 out of 26) of the children under ten, 38.5 per cent of those between ten and fifteen years, and 29.6 per cent of persons over fifteen were found to have been infected with bovine tubercle bacilli. Mitchell[2] states that 90 per cent of the cases of cervical gland tuberculosis in Edinburgh children under twelve investigated by him were due to bovine bacilli.

Cobbett,[3] after careful study of available evidence, arrives at the conclusion that "we do not yet possess the evidence which will enable a final verdict to be pronounced" as to the significance of bovine infection in human tuberculosis. One thing is, however, certain: In adults fatal bovine infection, if it does occur at all, is so rare that it is of no significance from any standpoint. Indeed, only in children under five years of age are bacilli of bovine origin apt to cause disease.

Bovine tubercle bacilli, when found in humans, are always "zoögenetic." If they were "anthropogenetic"—transmitted from man to man—the proportion of humans with bovine infections would be about the same in all organs. Bushnell[4] suggests that infection with human bacilli is more likely to occur because of sufficiency of opportunities through contact with tuberculous persons, and for this reason it occurs early in life. The child is thus immunized against infection with bovine bacilli. If this immunization were not so widespread, infection with bovine bacilli would be more common.

Virulence of Bovine Bacilli in Human Beings.—There appears to be some good and valid evidence to the effect that when a human being is infected with bovine tubercle bacilli, the disease produced is likely

[1] Lancet, 1915, 1, 1275.
[2] British Med., Jour., 1914, 1, 125.
[3] The Causes of Tuberculosis, London, 1917, p. 657.
[4] Epidemiology of Tuberculosis, p. 180.

to run a favorable, and even a benign, course; only rarely is death caused by these microörganisms. We know that it is the pulmonary form of tuberculosis which is fatal; while tuberculosis of the glands, joints, and bones tends to recovery in the vast majority of cases. Similarly, tuberculosis of the serous membranes, notably the peritoneum, often shows a tendency to recovery. We will see later on in this book that this is also true, to a certain extent, of the pleura. The meninges are an exception, for obvious reasons. Now, the peritoneum is very frequently affected by bovine tubercle. Moreover, tuberculosis of the cervical and thoracic glands is very common among children, yet the tuberculosis mortality among them is very low. This disease, in its various forms, kills less between three and twelve than at any other age period. Moreover, it has been found that in many instances caseous tissue obtained from tuberculous glands, while showing the presence of acid-fast rods, fails to infect animals when they are inoculated. It has thus been suggested that these mild bovine infections of the cervical, mesenteric and thoracic glands, while in themselves harmless, nevertheless confer immunity to the organism which may last for life, and for that reason adults are safe against infection by human tubercle bacilli. We shall revert to this point later on.

POISONS PRODUCED BY THE TUBERCLE BACILLI.

When tubercle bacilli enter the human body they do harm in various ways. Locally, they destroy the tissues in which they have settled, producing coagulation necrosis, etc., which will be discussed later on. By their proliferation they also produce general disturbances in the functions of the invaded body which can only be explained as caused by some poison liberated by the bacilli. The nature of these poisons is obscure at present, although strong efforts have been made to ascertain all the facts in this respect.

When dead tubercle bacilli are injected subcutaneously into the healthy animal, a distinct inflammation is produced at the site of the inoculation, frequently followed by suppuration. It is immaterial whether the bacteria have been killed by chemicals or by heat, the result is the same in either case. When dead tubercle bacilli are injected intravenously into rabbits, provided a sufficient quantity is employed for the purpose, a proliferation of tissue in the lung is produced similar to that of tubercle, containing, as it does, giant cells which may caseate. After intratracheal insufflation, tuberculous nodules with epithelioid and giant cells are produced.

On the other hand, when fluids containing the products of the metabolism of tubercle bacilli are injected in very large doses into normal and healthy animals, no toxic effects are produced.

These and other facts tend to show that the effects of the bacilli on the animal body are not due to mechanical irritation produced

at the site of the inoculation, but are the result of the liberation of toxic matter which acts both locally, producing coagulation necrosis, and generally, producing fever, etc. We know this, but *attempts to isolate a true toxin from tubercle bacilli have utterly failed, and with the intensive studies that have been made during the past thirty-five years along these lines, we have not yet been able to clearly define the tuberculous poisons.* They appear to be part and parcel of the living protoplasm of the tubercle bacilli, and are liberated only after the latter have been destroyed. In other words, *the tubercle bacilli belong to a group of microörganisms which do not secrete soluble toxins,* but nevertheless produce general effects on the body which they invade; their deleterious effects are the result of the action of *endotoxins.*

Tuberculin.—Koch was the first to discover that when dead tubercle bacilli are injected in large quantities into *tuberculous* animals, death is caused; when small doses are injected, only a slight reaction is caused at the site of the inoculation, which soon heals. On repeated inoculations he observed improvement in the condition of the sick animal. On these experimental findings he based his suggestion for the use of tuberculin as a diagnostic and therapeutic agent in tuberculosis.

Tuberculin consists mainly of the culture fluid in which the bacilli have grown, of disintegrated bacilli, or extracts of their protoplasm, or both. As originally prepared by Koch, the following process is pursued:

Tubercle bacilli are cultivated on bouillon made from fresh veal to which 1 per cent of dried peptone, 0.5 per cent of sodium chloride, and 5 per cent of glycerin are added. Within six to eight weeks of luxuriant growth at 38° C. the culture is poured into an evaporating dish, placed on a water bath, and evaporated to one-tenth the original volume, and any remains of bacilli are removed by filtration; containing 50 per cent of glycerin, the resulting preparation is quite stable.

It is thus clear that tuberculin is not a true toxin, nor is it a pure endotoxin; but a 50 per cent glycerin solution of the products of macerated tubercle bacilli in the culture fluid which are not destroyed by heat, and also any portion of bacilli which remains in the solution, or both.

Ever since the introduction of this original tuberculin, many other methods of preparation have been devised by Koch himself and others, but all have shown that the active principle is practically the same.

The Action of Tuberculin.—There are differences of opinion as to whether tuberculin depends in its action on a certain chemical principle, or on several chemical substances. In fact, the chemical composition of this preparation is obscure. Some have suggested that the active principle is a protein, or albumose. Klebs, Levene, and others believe that they have isolated various active principles; some have even obtained typical tuberculin reactions with these substances.

But, as will be shown when discussing the tuberculin reaction, any protein inoculated into a tuberculous individual produces practically the same effects—tuberculosis being invariably accompanied by an altered reactivity to these substances. It can be said emphatically that, at the present state of our knowledge, we are in the dark as to the active principle of tuberculin.

Healthy animals bear the injection of tuberculin in large doses without any harm; the same is true of healthy human beings. Koch injected into his own body 0.25 c c of tuberculin and suffered from a severe reaction; after his death an autopsy showed that he had suffered from extensive pulmonary tuberculosis. On the other hand, Hamburger administered as much as 500 mgs. of tuberculin into non-tuberculous infants and children without producing the slightest local, or general, reaction. Clinical experience among human beings, as well as in cattle—in which it is easy and feasible to determine by autopsy whether there are tuberculous lesions—has shown that a reaction after a large dose of tuberculin in an apparently healthy person is conclusive proof of an existing tuberculous lesion somewhere in the body. We shall show later on that this is true of the vast majority of people in civilized communities, and therefore reactions to large doses of tuberculin are of very little value to the clinician who looks for active tuberculosis.

The reason why tuberculin is harmless in healthy organisms, and produces such a pronounced reaction when injected into tuberculous organisms, is not clear. Various theories have been advanced to explain it. The most widely accepted explanation is that of Wolff-Eisner. He assumes that tuberculous infection produces specific antibodies in the tissues which break down the tuberculin molecule, just as the digestive enzymes break down certain albumin molecules producing innocuous, and highly poisonous, albumoses. The antibody which acts in this manner he calls tuberculolysin. In non-tuberculous organisms there is no tuberculolysin, and when tuberculin is injected it circulates within the juices, producing no toxic effects, and is finally eliminated, like other harmless foreign proteins. In the tuberculous organism the tuberculin comes in contact with the lysin, is broken up, and liberates a toxic substance which produces the reaction.

Phenomena of Hypersensitiveness.—When a rabbit is infected with tubercle bacilli, and four weeks later 0.1 to 0.3 c c of tuberculin is injected subcutaneously, the animal succumbs within six to twenty-four hours. Koch found that in animals infected eight to ten weeks previously 0.01 c c of tuberculin is sufficient to cause death. Injections of very small doses into tuberculous animals produce only a more or less severe reaction—fever, loss of weight, etc. This is obtained with injections of either living or dead tubercle bacilli.

When repeated small doses of tuberculin are injected, certain phenomena are observed which are not unlike those obtained after the injection of other foreign protein substances into an animal.

The tuberculin reaction is evidently a manifestation of tuberculo-protein hypersensitiveness. Some authors have, indeed, been inclined to ascribe the reaction to tuberculin to the action of the non-specific substances, glycerin, proteins, extractives, etc., contained in the tuberculin, and have argued that the reactions to repeated inoculations are anaphylactic phenomena. Perhaps the fact that the usual dose of tuberculin does not contain enough of foreign proteins disproves this contention, and shows that there must be some specific substances which are active in this regard. But this has not been proved conclusively.

Theoretically, it would be expected that tuberculin, provoking the same phenomena in the animal body as the living tubercle bacilli, should also have an immunizing effect. But, so far, nobody has been successful in an attempt at immunization of the body with dead tubercle bacilli, or any part of the culture in which they grow. More satisfactory results have been obtained infecting animals with living bacilli.

Tuberculin hypersensitiveness differs from anaphylaxis by the fact that in normal animals tuberculin may be injected in large or small amounts, at long or short intervals, without producing hypersensitiveness, and attempts at passive transference of tuberculin hypersensitiveness have led to doubtful results. Baldwin has been unable to produce transference, or passive anaphylaxis, from tuberculous guinea-pigs to healthy ones, and also from rabbit to rabbit, and from rabbit to guinea-pig. From human to guinea-pig the results were very doubtful, but to rabbit, partly successful. But another difference between anaphylactic shock and tuberculin hypersensitiveness may be mentioned. The former phenomenon appears immediately after an injection, while in the latter they are delayed for many hours; in the former there is a marked reduction in the temperature, etc., while in the latter the contrary is true.

Specificity of the Tuberculin Reaction.—We have seen that tuberculin produces obvious effects only in the infected organism. The question then arises whether the reaction it produces is strictly specific. Many workers have found that tuberculous animals react to, and may even be killed by, the injection of any foreign bacterial protein of non-tuberculous origin in the same manner as by tuberculin. In human beings there was also found hypersensitiveness to non-tuberculous extracts from bacilli closely resembling the hypersensitiveness induced by tuberculin. Even the cutaneous tuberculin reaction can be produced by non-tuberculous toxins inoculated in the same manner as tuberculin is applied in the von Pirquet and other tests.

The changes in reactivity to tuberculin may be induced by non-tuberculous proteins and toxins. The general reaction, the fever, with concomitant subjective symptoms, such as headache, anorexia, etc., also the local reaction at the site of the inoculation, and finally even the so-called "focal reaction" manifesting itself in the tuberculous

lesion, have all been produced by non-tuberculous substances. More recently R. Schmidt[1] found that parenteral milk injections are often followed in tuberculous subjects by general, and focal reactions which cannot be distinguished from those produced by tuberculin. On the other hand, tuberculin has been known to produce these reactions in patients suffering from leprosy, syphilis, etc. The suggestion that this does not militate against the specificity of the tuberculin reaction, because these diseases may be combined with tuberculosis, does not explain every case.

It has also been found by Mettetal,[2] and others, that individuals who react to tuberculin also react in almost the same fashion to saline solutions, which would indicate that it is not necessarily the specific bodies in the tuberculin which are responsible for the fever, malaise, etc. At any rate, tuberculin is not the only substance that produces these phenomena in tuberculous individuals.

Autopsy control has not cleared up the problem. There have been reported cases in which a positive reaction was obtained during life, but no tuberculous lesions could be discovered on careful dissection of the body after death, and the reverse. In cattle it was found that only 85 to 90 per cent of those reacting to tuberculin show tuberculous changes on dissection after slaughter, while 10 per cent of those which do not react show tuberculous changes in some organs. These facts have important bearings on the problems presented by tuberculin as a diagnostic agent and will be more fully discussed later on.

Another problem arises when changed reactivity to tuberculin is found. Does it invariably indicate that the body is at the time harboring living and virulent tubercle bacilli? Do individuals who have at one time passed through a tuberculous infection, but in whom the lesion has completely cicatrized, also show hypersensitiveness to tuberculin? To the first question we have a positive answer: Many healed, cicatrized, and calcified tuberculous lesions have been found to harbor virulent bacilli, as has been proved experimentally. These bacilli are, in fact, responsible for acute exacerbations observed in quiescent and latent tuberculosis; they may also be held responsible for the onset of the average case of phthisis in adults, as will be shown elsewhere. But what is of more importance is whether, once acquired, the tuberculin hypersensitiveness remains throughout the life of the individual. This is a problem which has not yet been investigated to an extent as to warrant a positive answer.

Outside of such theoretical considerations, these problems have great practical bearings on the utility of tuberculin as a diagnostic agent, which is discussed in Chapter XIX.

[1] Deutsch. Arch. f. klin. Med., 1920, **131**, 1.

[2] Valeur de la tuberculine dans la diagnostic de la tuberculose de la première enfance, Thèse de Paris, 1900.

CHAPTER II.

TUBERCULOUS INFECTION.

The Problems of Infection.—With the discovery of the tubercle bacillus in 1882 it was at once concluded that practically all the problems of phthisiogenesis had been settled. The infective agent, the bacillus, enters the human body, implants itself in some tissue; by its growth and metabolic processes it produces toxic symptoms and, causing caseation and liquefaction, destroys vital organs, etc. With this knowledge, it was thought that the prevention of the disease had been reduced to simple principles: The destruction of the bacilli wherever found, and the prevention of their entry into the human body, when attempts at their destruction fail for any reason.

To destroy the bacilli it was necessary to ascertain all the places where they are found in Nature. This was apparently an easy matter. We know that the tubercle bacillus is a strict parasite, living and multiplying only in the human and animal body. Investigations by Sander tend to show that, within certain limits, they can proliferate on vegetable media during the hot summer months, but it is problematical whether this mode of life explains any infection in man. After the facts gathered by careful investigators are taken into consideration, there is no doubt that the only suitable soil for life, growth and multiplication of this bacillus is the animal body, and that the secretions and excretions of diseased persons and animals are the only means of disseminating the disease.

We have shown that bacteriologists have distinguished at least four main types of pathogenic tubercle bacilli: the human, the bovine, the avian, and the reptilian. Practical experience has shown that the last two types, those of birds, and of cold-blooded animals, play no rôle in the epidemiology of tuberculosis in human beings, at least not a very significant rôle. There are consequently left the human and bovine types to be considered as etiologically important in tuberculosis in human beings.

Careful investigations by Theobald Smith, William H. Park, A. S. Griffith, Fraser, The British Royal Commission, The German Imperial Health Board, and others, have shown that more than 99 per cent of phthisis in adults, and about 85 to 90 per cent of serious tuberculous disease in children are due to the human type of bacillus; that the bovine type is found in about 10 per cent of tuberculosis in children, and in pulmonary tuberculosis in adults this type is so exceptional as to make each case worthy of careful reporting. It also appears

from the evidence thus far gathered that tuberculosis in children due to bovine bacilli is mostly of the milder forms of the disease—surgical tuberculosis, of the glandular systems, especially of the thoracic and the abdominal glands, of the joints, bones, and skin. In other words, the diseases caused by the ingestion of bacilli with milk from tuberculous cows are not of great significance, except perhaps in infants, when compared with the immensity of the problems presented by infections with the human type of bacilli, causing phthisis in adults, and most cases of fatal tuberculosis in infants.

For these reasons, some authors have stated that bovine infections may be disregarded; only infection with bacilli acquired through the entry of tubercle bacilli which have been incubated, so to say, in tuberculous human beings, is to be combated, if phthisis is to be eradicated at all. The corollary to be drawn is that the sources of the tubercle bacilli are mainly human consumptives.

Mutation of the Types of Bacilli.—Further study has, however, complicated this problem. It has been suggested by many authors, notably Orth,[1] Rabinowitsch, Beitzke, Much, and others, that bovine bacilli, remaining in the human body for a long time, and adapting themselves to the surroundings, may acquire the characteristics of the human type, a kind of biological transformation of type, or mutation. It is clear that in our attempts at eradication of phthisis this problem is of immense importance. The 10 per cent, or more, of children in civilized countries who are infected during childhood with milder forms of tuberculosis thus harbor the bovine bacilli within their bodies for many years, during which time they adapt themselves to the surroundings within the human body, and when they cause phthisis in the adult we find them with the characteristics of the human type.

In support of this assertion it was shown that very often "atypical" bacilli are found in cases of tuberculosis; they are microörganisms which cannot be classed with either the human, or the bovine type. They have been called "transitional" types; types which may have been originally bovine, but after sojourning in the human body for some time, are on the way to acquiring traits of human bacilli.

The British Royal Commission says in this connection that they "are inclined to regard transmutation of the bacillary type as exceedingly difficult, if not impracticable, of accomplishment by laboratory procedure; though in view of certain instances in which we obtained from one and the same human body both types of bacillus, we are not prepared to deny that transmutation of one type into another may occur in Nature." "Direct experiment has not succeeded in proving that a tubercle bacillus of given type can be transformed into one of another type by being made to reside in the body of a new host in which tuberculosis, when it occurs naturally, is caused by the latter type of bacillus," says Cobbett.[2] Arloing, Marcus, Rabinowitsch,

[1] Drei Vorträge über Tuberkulose, Berlin, 1913.

[2] The Causes of Tuberculosis, London, 1917, p. 368.

Sorgo, Musemeier, Dammann, and especially Löwenstein,[1] claim to have been able to produce changes in the morphological and cultural characters, and in the virulence of bacilli by passage through various animals, or cultivating them in different media. But Park and Krumwiede[2] say: "We have carefully examined the reports of numerous workers on this point, and cannot admit that the evidence for the transformation of type is complete." Theobald Smith, after studying the evidence, also arrives at the conclusion that "in general the results of these passages have been negative, so far as any recognizable modification of type is concerned." Park's suggestion that the change in type observed after passing through a series of animals is due to additional bovine infection has a great deal in its favor. As has been shown by Cobbett,[3] the more the conditions for carrying out such researches are made to approach the ideal, the rarer become the instances of apparent modification of type. Cases in which both types were found in human beings have been reported.

We are therefore justified in concluding with Park and Krumwiede that "the two types are probably different due to residence in different hosts over long periods of time, and as such are stable. The evidence of rapid change is incomplete and inconclusive." In the human disease the stability of type is apparently beyond question. Some cases have been followed for long years and the type of the bacillus has been found to be unaltered. Weber and Steffenhagan have followed for ten and a half years a case of surgical tuberculosis and always found bovine bacilli, without changing their typical characteristics.

However, the weight of evidence is in agreement with Cobbett[4] to the effect that if transformation of type does not occur in our laboratory experiments which, prolong them how we will, are necessarily limited in time, it does not follow that an exceedingly slow modification of type does not take place when a suitable change of host occurs, as for example when bovine tubercle bacilli take up their residence for several generations in man, pig, or horse. Such a change is perhaps dimly indicated in some of the experiments with viruses of the bovine type taken from these species. This slow alteration, which appears probable (though the actual evidence for its existence is very slender) is, if it occurs at all, of a magnitude altogether different from that of the more or less sudden and complete changes of type which have appeared in some of the passage experiments. But such slow changes hinted at here are of little more than theoretical importance.

On the other hand, Brownlee,[5] taking into consideration that the frequency with which bovine tuberculosis occurs decreases with age

[1] Vorlesungen über Tuberkulose, Jena, 1920, p. 80.

[2] Tr. Sixth Ann. Meet. Nat. Assn., Study and Prevent. Tuberc., 1910, p. 322; Jour. Med. Research, 1911, **20**, 313; 1912, **22**, 109.

[3] Loc. cit., p. 367.

[4] Ibid., p. 369.

[5] An Investigation into the Epidemiology of Phthisis in Great Britain and Ireland, Part III, London, 1920, p. 56.

(see p. 33), is inclined to the opinion that "the evidence strongly suggests that in the human laboratory a change from the bovine to the human type takes place. This is rendered more probable by the fact that when bovine bacilli have been injected into horses and dogs changes in the character of the organisms associated with the loss of some of the characteristics peculiar to the bovine type of organisms have been observed." He also suggests the possibility that bovine and human tubercle may be modifications in two directions of an organism standing somewhere between. It is also possible that the organism may have an independent existence, that is to say, may live saprophytically outside the body under conditions not at present understood. A well known example of this kind is the streptococcus, which is a notoriously delicate organism when grown under laboratory conditions, but which is ubiquitous.

The weight of evidence is, however, in favor of human phthisis being due almost exclusively to human bacilli, and that infection during childhood with bovine bacilli cannot be held responsible for phthisis in the adult, because, so far, it has not been proved that mutation of one type into another takes place.

The source of the bacilli causing phthisis in the adult, and serious or fatal tuberculosis in infants or children, appears to be the tuberculous man who expectorates myriads of bacilli fit for entering healthy persons and causing disease.

The Channels of Entry of the Tubercle Bacilli.—In our attempts at prevention of tuberculous disease we must be first definitely informed on the channels through which these microörganisms enter the human body. To the average person, lay or medical, who has informed himself from current popular literature, this question has been answered satisfactorily. If the bacilli are derived from human sources, they have usually been inhaled, and exceptionally ingested; if from bovine sources, they have been ingested.

But it may be stated without fear of meeting contradiction from competent sources that this problem has not yet been solved to the satisfaction of all who are entitled to an opinion. Römer,[1] one of the most active experimental workers in the field of tuberculosis, and one of the best qualified to speak authoritatively, says that *none of the given channels of entry of the tubercle bacilli is alone sufficient to adequately solve all the problems presented by tuberculous infection.*

Obviously there are four portals of entry which are to be mentioned as possible:

1. *Inoculation* into the skin or mucous membranes.
2. *Inhalation* through the respiratory passages, or aërogenous infection.
3. *Ingestion* through the digestive tract, intestinal infection.
4. *Germinative, or placental infection,* the bacilli being derived from the parents before the birth of the individual.

[1] Handb. d. Tuberkul., 1, 247.

Inoculation and Contact Infection.—Considering the ubiquity of the tubercle bacilli, the skin should be the most common channel of entry of these germs into the body. The transfer of germs from hand to hand, and from hand to mouth, is very difficult to avoid when it is borne in mind that every individual living in a civilized community must come in contact with tubercle bacilli in his daily routine life. Palmer[1] enumerated the chances of acquiring a communicable disease by contact and he mentions one hundred and nineteen points of contact, from touching of door-knobs, faucets, various eating utensils, and money, to handshaking, receiving newspapers, touching telephone directories, saliva-moistened street car transfers, etc. J. B. Rogers[2] found experimentally that such objects as gauze used to cover the mouth when coughing, pillow cases used twenty-four hours, patient's hand, spoons used by patients, magazine covers picked up indiscriminately from the wards, and door-knobs frequently handled by patients, are contaminated by virulent tubercle bacilli. On the other hand, other workers along these lines have found that the dangers of infection in this manner are rather remote.[3]

Inoculation of tubercle bacilli into the skin and mucous membranes may cause disease. This has been proved beyond any doubt experimentally and clinically. Inoculated tuberculosis is most virulent on virgin soil, as is observed in animal experimentation, and in infants who have thus been infected during ritual circumcision as practiced by orthodox Jews, of which many cases have been reported, notably by Arluck, Bernhardt, Winocouroff, Holt, and others. Local tuberculous lesions have also been observed to result from infections acquired by surgeons, anatomists, pathologists, veterinarians, butchers, etc. But there are more curious ways in which this mode of infection has been observed to occur. Deneke, Moro, and others, report cases in which children have been inoculated with tubercle bacilli while breaking sputum receptacles. There have also been reported infections acquired while tattooing (Ernst), while piercing the ears in girls, the needle being moistened with the saliva of a tuberculous individual (Grosser, Grossmann, Patzold), by hypodermic needles, etc.

Inoculation of tubercle bacilli into susceptible animals, and human infants, produces a fatal bacteremia, as a rule, but in adults only a local lesion at the point of entry of the germs results. The reasons for these differences will be discussed later on. On the other hand, when considering cutaneous skin affections, such as the various clinical forms of lupus, or the so-called tuberculides, it must not be hastily concluded that they are invariably acquired by inoculation. As will be shown later on, while discussing hematogenous and lymphogenous infection, the germs are usually brought there by the blood or lymph stream.

[1] Am. Jour. Pub. Health, 1919, **9**, 267.
[2] Am. Jour. Pub. Health, 1920, **10**, 345; Jour. Am. Med. Assn., 1920, **75**, 1690.
[3] See Brown, Petroff and Pasquera: Am. Rev. of Tuberculosis, 1919, **3**, 621.

Immunity of the Skin.—Contact infection, even though it may occur, is not of great importance in phthisiogenesis. Of all the organs, the skin appears to be the least vulnerable to tuberculosis, as has been shown by Lewandowsky.[1] Its low temperature, as well as its chemical and anatomical structure appear to offer unfavorable conditions for the growth and proliferation of tubercle bacilli; in fact, there is strong evidence that it offers vigorous resistance to tuberculous infection. Facts gathered from comparative pathology show that animals known to be very susceptible to tuberculosis have only rarely skin lesions. Tubercle bacilli may penetrate the normal animal skin and produce visceral tuberculosis without leaving any traces on the skin itself. Although Takeya and Dold state that they could always find tuberculous changes at the point of entry, C. Fränkel, Courmont and André, Lesieur, Babes, and others, report the contrary. Babes and Fränkel found tubercle bacilli in the lymph vessels of the subcutaneous tissues four to forty-eight hours after rubbing the germs into the skin of animals, which shows clearly that they may penetrate the skin without damaging it.

The immunity of the human skin is also evident from the fact that, though exposed to infection more than any other organ, it is but rarely tuberculous. Indeed, tuberculous skin diseases are very rare in phthisical patients who can hardly avoid inoculation. It is the consensus of opinion that the various tuberculides seen in tuberculous patients, especially children, such as the papulo-necrotic tuberculides, lichen scrofulosorum, erythema induratum, sarcoid, etc., are manifestations of reactions of skin hypersensitiveness of tuberculous individuals, brought about by dead bacilli carried by the blood and lymph streams. Other tuberculous skin diseases, such as the various forms of lupus, etc., even when due to local infection, as is the case with tuberculosis verrucosa cutis, etc., may disfigure, but they nevertheless run a mild course, hardly ever leading to fatal termination by extension of the disease to visceral organs. Moreover, the skin lesions themselves remain localized for many years without showing any pronounced tendency to extension. When compared with the skin lesions of tertiary syphilis, which tend to spread and destroy, it is clear that tuberculosis of the skin is a rather mild disease. "The skin is not a culture medium for the tubercle bacillus, it does not permit it to grow and proliferate," says Lewandowsky. Microscopically, this is confirmed by the paucity of tubercle bacilli in most tuberculous lesions of the skin. It must also be mentioned in this connection that various investigators have found that a large proportion of skin lesions are caused by bovine tubercle bacilli, which were isolated in pure culture from lesions of certain forms of lupus vulgaris.

Available evidence thus tends to show that infection through the skin is hardly of any importance in the etiology of tuberculosis of

[1] Die Tuberkulose der Haut, Berlin, 1916, p. 32.

visceral organs, notably the lungs. As will be shown later on, even in cases in which a tuberculous skin disease terminates fatally with symptoms of miliary tuberculosis, it is usually due to a reactivation of dormant lesions in the glands, or lungs.

Infection by Inhalation of the Bacilli.—That the virus of tuberculosis is inhaled with the inspired air has been asserted for centuries by physicians, and Villemin suggested this mode of infection after his experimental investigations. But Koch and his pupil Cornet[1] were the first to prove that dust containing tubercle bacilli derived from desiccated sputum is highly infectious to guinea-pigs. Cornet's experiment with dried sputum scattered over a carpet on which the animals were compelled to live while the carpet was often swept with a stiff broom, has remained classical, and is often quoted as proving conclusively the dangers lurking in dried sputum in the average dwelling inhabited by careless consumptives. On the basis of such experiments rested the entire inhalation hypothesis of tuberculous infection.

The fact that diffuse daylight, especially sun-rays, kills tubercle bacilli, and soon renders them avirulent, would largely exclude infection through sputum deposited in the street and in large, bright sunny rooms. But the average consumptive, derived as he is from the poorer strata of population, and living in a squalid dwelling, lacking sufficient light, may deposit sputum which retains its virulence for a long time.

Many valid objections have been raised against the theory that desiccated tuberculous sputum is the main source of infection in man. Flügge,[2] and many others, have shown that in the ordinary course of human events things are not as simple as stated by Cornet and Koch. The experiments with the carpet are not altogether analogous to the conditions found in human dwellings, and by no means prove that infection is acquired mainly through the inhalation of dust laden with dried tuberculous sputum. Such large quantities of sputum as were used by Cornet in his experiments on guinea-pigs are exceedingly rarely, if ever, found in the most squalid of dwellings. It is also doubtful whether dust laden with virulent tubercle bacilli is often raised to the height of the human head to be inhaled in sufficient amount to infect, even while the floor is being swept.

In fact, further investigations by Flügge, Neisser, Köhlisch, and others have not yielded the same results as those reported by Koch, Cornet, and their followers. It was found that in houses inhabited by consumptives the sputum deposited on the floors is not often perfectly dried and thinly pulverized, capable of rising with the dust to the height of five or more feet from the ground. Moreover, conditions in unsanitary homes are, as a rule, not conducive to drying the sputum soon after it has been eliminated by the consumptive. And if it takes time to dry, it must be remembered that the bacilli lose their virulence within ten days, owing to putrefactive pro-

[1] Verhandl. Berl. med. Gesellsch., 1899, **30**, 91.
[2] Ztschr. f. Hyg. u. Infectionskrankh., 1909, **30**, 107.

cesses on the floors of filthy houses, and the diffuse light which acts during the day, or artificially, during the night. It is also noteworthy in this connection that in the average house there are no air currents strong enough to raise the dust to the height of about five feet.

It may seem incredible, yet it is a fact that it is exceedingly rare to find a house where proper precautions are taken as to expectoration in which the collected dust shows virulent tubercle bacilli. Even in houses inhabited or frequented by consumptives—sanatoriums, dispensaries, railroad stations, factories, cars, etc.—no dust containing virulent tubercle bacilli has been found in most cases investigated. Thus, Köhlisch[1] could not infect guinea-pigs, which are very susceptible, with dust collected in houses inhabited by consumptives, and he concludes that it has no significance in the origin of tuberculous disease. Wagner collected dust in a sanatorium at Zurich, in such places in which the air stream could have dispersed it, and injected it intraperitoneally into guinea-pigs and found that in only 3.5 per cent of cases did infection take place. Brown, Petroff, and Pasquera report about dust collected by a vacuum cleaner from a large rug in the living room of the Trudeau Sanatorium. It proved negative when injected into a guinea-pig. The same result was obtained with the dust from a room in the infirmary occupied by a patient with numerous tubercle bacilli in the sputum and a cough so explosive that the mouth was rarely covered. They also collected dust before the daily cleaning by swabbing with sterile swabs the bed, tables, chairs, bed frames, corners of the rooms and walls near the patient. The swabs were washed in sterile broth, the washings treated with normal sodium hydroxide, incubated for half an hour, then neutralized with normal hydrochloric acid, centrifugalized, and the sediment divided into three portions. One was inoculated into gentian violet media, another stained on a slide for microscopic examination, and the third inoculated subcutaneously into the inguinal region of guinea-pigs, two for each swab. In all twenty-four animals were used. All the slides were negative as regards tubercle bacilli, and none of the twenty-four animals showed any signs of tuberculosis when carefully examined at the autopsy. The dust in the rooms inhabited by two tuberculous patients outside of the sanatorium was also found negative for tubercle bacilli. The same was true of the mouth pieces of the telephone used by patients at the sanatorium. Dust collected in the streets hardly ever shows the presence of living and virulent tubercle bacilli.

It thus appears that the hypothesis of pulmonary tuberculosis resulting from infection brought about by inhalation of bacilli entering the respiratory passages with the inhaled air during respiration is not conclusively proved. Those interested in this particular phase of tuberculous infection may find ample data in Calmette's[2] most recent work.

[1] Ztschr. f. Hyg. u. Infectionskrankh., 1916, **81**, 203.

[2] L'infection bacillaire et la tuberculose, Paris, 1920, pp. 127–144.

Droplet Infection.—It is obvious that though infection through the inhalation of dust containing desiccated tuberculous sputum is undoubtedly possible, this is not the only, or the most common, mode of spontaneous infection of human beings under "natural conditions," and many have maintained that in the vast majority of cases infection is accomplished directly from one person to another. The moist droplets eliminated by consumptives while speaking, and especially while coughing and sneezing, may be inhaled by persons who happen to be in their proximity. Flügge[1] and his followers, who have done considerable experimental work along these lines, are satisfied that under natural conditions the dissemination of tuberculosis from man to man, "droplet infection," is the most common mode.

Careful research has shown that the air exhaled by consumptives during ordinary and quiet breathing is free from tubercle bacilli, but the moist droplets eliminated from the mouth while talking, coughing, sneezing, etc., do often contain tubercle bacilli which may remain floating in the air for some time. Indeed, it has been found that the *Bacillus prodigiosus* may thus float in the air for five hours. In various parts of a large hall Laschtschenko[2] placed Petri dishes containing culture media. He then washed his mouth with a suspension of *Bacillus prodigiosus*, a microörganism which is not found naturally in the air, and which may be easily identified. After delivering a speech, he proceeded to collect the dishes and placed them in an incubator. Many of the culture media in the dishes showed excellent growth of the bacteria. Gordon[3] repeated this experiment and obtained the same results.

The problems of droplet infection are many, and it may be said that no agreement has yet been arrived at by all who are entitled to an opinion. Experimentally it has been shown that when a tuberculous patient coughs at a cover-glass, bacilli are found on the slide in a variable proportion of cases. It seems that in order that the bacilli should be deposited on the cover-glass it must be held close to the mouth of the patient. Some authors found that while when held 40 to 80 cm. away from the patient's mouth 30 per cent of the cover glasses show tubercle bacilli microscopically; no bacilli are deposited when it is held one meter or more away from the mouth of the patient. Engelmann showed that when the slide is held at the side of the patient's face, negative results are obtained even when the distance is but 30 cm. from the patient's face. In this country J. B. Rogers obtained positive results at a distance of fifteen inches, but Brown, Petroff and Pasquera met with failure while making somewhat similar experiments.

Placing susceptible animals before consumptives is another experimental method. Flügge immobilized guinea-pigs and had tuberculous

[1] Die Verbreitungsweise und Bekämpfung der Tuberkulose auf Grund experimenteller Untersuchungen, Leipzig, 1908.

[2] Ztschr. f. Hyg., 1899, **30**, 125.

[3] Suppl. Ann. Report Med. Off. Loc. Govt. Board, 1902–3, p. 425.

patients cough directly into their mouths. The pigs became tuberculous in 6 out of 25 cases. Möller had tuberculous patients cough at guinea-pigs daily for several weeks, and in 2 out of 14 cases was tuberculosis thus transmitted.

These experiments were apparently more often positive than in the case of experimental infection with dust containing desiccated tuberculous sputum, and Flügge and his followers conclude that this mode of infection is the most important under natural conditions.

But even these experiments are open to question. The animals were held tightly for hours, directly exposed to the faces of the consumptives who coughed directly into their open mouths. Such exposure never occurs in human beings, except perhaps in cases of tuberculous mothers holding their crying babies on their arms, and coughing directly into their open mouths, which may be observed now and then among certain classes, but after all cannot be considered very common.

A somewhat analogous experiment among humans was made by F. Hamburger.[1] He placed a feeble-minded boy, aged nine years, in a room in which three tuberculous girls lived. Because of the mental state of the boy, the girls avoided him and never came into close contact with him. And he remained free from tuberculous infection, as shown by the negative tuberculin reaction, for seven months. On the other hand, in a room in which a tuberculous patient was harbored, four children, aged two, four, five and ten years respectively, were brought in. Within four weeks all the four children, who before coming into the room with the tuberculous patient were tuberculin negative, showed a positive reaction to tuberculin. A somewhat similar observation was made by Hess in an infant asylum in New York City where a tuberculous nurse thus infected several infants in a ward in which she was stationed.

Even conceding that droplet infection is an important mode of transmission of tuberculosis, it must be realized that it depends on many factors which are not always, or even often, operative. When a healthy person is at a distance of three feet from the coughing patient, the droplets will not reach far enough to become a possible infective agent, excepting perhaps when carried by air currents. Another important factor is the dose of the bacilli that may thus be inhaled. As has been shown elsewhere, small numbers of bacilli are easily taken care of by the human organism. It is also a fact that tubercle bacilli thus eliminated do not remain floating in the air for any length of time, but sink to the floor, where they are soon rendered innocuous, as was already mentioned.

It is thus obvious that only when contact with the consumptive is very close, intimate, and prolonged, which in ordinary life occurs, as a rule, only in mothers with suckling infants, or between husband

[1] Wien. klin. Wchnschr., 1919, **32**, 33.

and wife, droplet infection may become a serious menace. And even in these cases there are natural safeguards.

Considering the evidence thus far brought together at its face value, it appears that *inhalation of dust containing tuberculous sputum, or of droplets expelled by consumptives while talking, coughing, and sneezing, may infect a healthy person, yet the evidence that these are the most frequent modes of the dissemination of tuberculosis is inadequate.*

From time immemorial physicians have attributed the transmission of infectious diseases to the inhalation of the virus. To the ancients "infection" meant everything that contaminates the air (Infection, from the Latin *infectionem, infectus,* or more exactly impregnated). This has notably been the case with the endemic diseases of childhood, and for a long time yellow fever, typhoid, typhus, malaria, relapsing fever, bubonic plague, etc., were all considered inhalation diseases, and proofs were at hand to substantiate these contentions. Recently more exact studies have shown conclusively in some, and with a high degree of probability in others, that they are altogether transmitted through the agency of certain insects. Indeed, physicians of a few generations ago drew analogies between tuberculosis and malaria, typhus, etc., showing that they were all caused by the inhalation of the virus.

Natural Barriers against Inhalation Infection.—Notwithstanding the various disharmonies which may be found in the structure and functions of the human body, and which Metchnikoff has so cleverly enumerated in one of his books, the respiratory tract is provided with a most wonderful protective apparatus for the prevention of the entry and implantation of bacilli in the deeper respiratory passages. Indeed, no organ in the body, excepting the central nervous system, is fitted out with better safeguards in this regard.

The bacilli cannot enter the lungs with ease. The nasal passages, mouth and throat act as excellent filters, detaining the inhaled dust. Even when some microörganisms in the inhaled air pass all the barriers, the mucus secreted all along the tract, the ciliated epithelium, etc., soon remove them as foreign bodies, when necessary assisted by cough, which has the function of clearing the lungs. Investigations of normal lungs have shown that they are germ-free; animal inoculations made by F. Müller, Goebel, Klipstein, Bartel, and others, have failed to reveal any pathogenic microörganisms, though Dürck found the contrary to be true. But in such cases it has been suggested that the germs reached the lungs shortly before death and could not be expelled, or destroyed by the dying tissues. The few bacteria which may remain within for any reason are, under normal conditions, well cared for by the extensive lymphatic apparatus which surrounds all the bronchi and bloodvessels, even the terminal bronchioles, and takes up bacteria, destroying them or at least rendering them innocuous.

From animal experiments, conducted for years, Bacmeister[1] shows

[1] Die Entstehung der menschlichen Lungenphthise, Berlin, 1914.

that while tubercle bacilli are rarely found in the lungs of animals compelled to inhale dust containing the germs, he never observed that infection of the normal lung was caused in this manner, and he concludes that the bacilli must be hindered in their development, destroyed, or carried away from the lungs by the lymph and blood stream. There is no reason against the assumption that the normal human lung acts in the same manner, and that bacilli which may succeed in penetrating into deep air vesicles are removed or destroyed before they can gain a foothold and cause disease.

It must, however, be borne in mind that dust of any kind may, and at times does, reach the lungs with the inspired air, as is evident from the large number of cases of pneumokoniosis of various degrees. Tubercle bacilli may likewise be brought there with the inspired air or with dust. But whether they cause disease in every case in which they reach the lungs is a disputed problem, the weight of evidence being against such a contention. Indeed, it has been proved that tubercle bacilli may remain alive and virulent in the tracheobronchial glands for years without causing disease, or even changes in the glands. Investigations by Bartel and Weichselbaum, Harbitz, and others, have shown that this is frequently the case, and it explains the latency of tuberculosis in many cases (see page 143).

That tubercle bacilli on mucous membranes do not invariably cause disease is proved by another fact. These microörganisms have been found on the mucous membranes of the nose, throat, and mouth of healthy individuals. Noble W. Jones[1] found them in the nasal cavities of healthy persons in the ordinary walks of life, especially those who cared for consumptive patients. Strauss[2] found tubercle bacilli in the nasal cavities of healthy individuals living in houses inhabited by phthisical patients. Alexander[3] found them in very large numbers on the mucous membranes of patients suffering from ozena, but who had no symptoms or signs of tuberculosis. These facts, taken in connection with the fact that tuberculosis of mucous membranes of the pharynx, nose, and mouth is exceedingly rare even in consumptives, show that these structures possess a certain natural resistance against tuberculosis. That it is not solely due to the immunity acquired by previous tuberculous infection is shown by the fact that, as a primary infection, tuberculosis of these parts is exceedingly rare, though it must be admitted that while entering the body, by inhalation or ingestion, the bacilli must pass them and they have greater opportunities to implant themselves there than in the deeper respiratory passages.

A lymphatic apparatus of normal structure and function evidently insures against the implantation and pathogenic action of all kinds of bacteria in the respiratory passages. Otherwise we would all succumb

[1] Med. Record, 1900, **58**, 285.
[2] Bull. de l'Acad. de méd., Paris, 1894, **32**, 18.
[3] Berl. klin. Wchnschr., 1903, **40**, 508.

to various microbic diseases, including tuberculosis. It is only when the natural protective forces fail that tuberculous disease may be caused in this manner.

On the other hand, it must be emphasized that the lungs are very much exposed to infection from the blood stream, and hematogenous infection may easily localize itself in these organs. The lungs are the first filter for everything that may be carried by the venous circulation. When the lymphatic apparatus is injured by anthracosis, which is very frequent to some degree in nearly all adult city dwellers, it is not capable of removing tubercle bacilli which may be brought to it with the blood stream. But here, again, it appears that persons suffering from anthracosis, with but few exceptions, are no more liable to tuberculosis than others (see p. 132). In fact, coal miners are relatively immune.

Difficulties in the Way of Establishing the Portals of Entry of Tubercle Bacilli.—The reasons why experimental investigations have failed to adequately solve the problems of the aërogenous etiology of phthisis are evident when we bear in mind that *pulmonary tuberculosis, as met with in human beings, showing isolated foci which extend slowly downward in the lungs, never occurs spontaneously in animals; nor has it ever been induced artificially, or experimentally, in animals.*

Really active initial lesions in the human lungs have only rarely been encountered at necropsies. Most cases examined on the autopsy table are advanced, and it is very difficult, or impossible, to decide which was the initial lesion. Even the initial lesions, found in individuals who died from causes other than tuberculosis, and reported by Schmorl,[1] Birch-Hirschfeld,[2] Lubarsch,[3] Beitzke,[4] and others, have not cleared up definitely the problem whether the bacilli were brought to the site of the lesion by the inspired air or the blood stream. It has, however, been found that even at that stage both the bronchioles and the bloodvessels were affected to such an extent that either, or both, could be considered the portal of entry. It is difficult or impossible to decide which is the initial lesion, even in experimental tuberculosis. "The fixing of the portals by the so-called oldest lesion," says Ravenel,[5] "is open to serious question. I have produced fatal pulmonary tuberculosis in monkeys by feeding, with very insignificant intestinal lesions. All the oldest lesions were located in the lungs and bronchial glands, yet the method of feeding largely precluded the possibility of the tubercle bacilli reaching the lung, except through the digestive tract." This is well known to every observing clinician and pathologist. It is not the rule that the most extensive, perhaps the fatal, lesion was the first. Indeed, in most cases we find a healed or

[1] München. med. Wchnschr., 1902, **49**, 1379.
[2] Deutsch. Arch. f. klin. Med., 1899, **44**, 58.
[3] Virchow's Arch., 1913, **213**.
[4] Berl. klin. Wchnschr., 1909, **46**, 388.
[5] Jour. Am. Med. Assn., 1916, **46**, 613.

smouldering lesion in one lung, while the newer lesion, in the opposite lung, is progressive and destructive. Similarly, we meet with mild, perhaps almost healed, lesions in the lungs, while a secondary lesion in the kidneys, or some other visceral organ, is the most important in causing a fatal termination. We are not always sure in assigning the portal of infection to the small gland which is found to harbor an old tuberculous lesion.

The fact that the regional lymphatic glands and lymph nodes are usually implicated at an early stage points to a hematogenous localization, but it may also be explained by the aërogenous hypothesis.

It is obvious that the inhalation of the bacilli does not exclude hematogenous distribution and their final localization at some point distant from the point of entry. Ribbert, Bacmeister, Lubarsch, Ravenel, Theobald Smith, and others, have pointed out that microorganisms brought into the bronchial tree by the inspired air may pass through the mucous membrane into the lung tissue without producing a visible lesion at the point of entry; pass along the lymphatics into the regional lymph nodes and from there carried by the blood stream into the pulmonary apices. But that this is in all probability rare, may be assumed when it is recalled that only few bacilli can reach the bronchi, and of these but few are allowed to pass through the normal mucous membrane of these tubes and the alveoli, and they are usually rendered innocuous by the protective properties and functions of the lymph and blood, as was just shown.

Hematogenous Infection.—Many look at phthisis as hematogenous in origin: The tubercle bacilli are assumed to enter the body at any point, the respiratory or digestive tract, or even through the skin, and are carried by the blood stream until they reach a point where the tissues have a low power of resistance, an organ which offers a favorable soil for the growth and action of these microörganisms. Considering the enormous frequency of pulmonary phthisis, it is evident that in the vast majority of human beings the lungs offer a good breeding-point for the tubercle bacilli. The localization of the bacilli is thus accomplished in the same manner as their localization in joints, the peritoneum, the meninges, etc.—by the blood stream.

The hematogenous origin of phthisis is especially urged by Baumgarten, Ribbert, and Aufrecht. According to Baumgarten, tubercle bacilli in the inspired air may infect the mucous membranes of the upper respiratory tract whence they are carried by the lymphatics to the regional glands—the submaxillary, cervical, and supraclavicular, which are so often enlarged in tuberculous children. Entering the superior vena cava they may be carried by the blood stream to the lungs, causing typical interstitial tubercle of these organs and finally extend, while growing, to the alveolar walls, or within them. Aufrecht holds that the primary tuberculous lesion is always in the vascular walls, which are affected by bacilli brought to them by the blood stream. Through the veins they pass into the right heart; or from

tuberculous bronchial glands they get into the pulmonary artery or its branches, when the lymph channels are obliterated by inflammatory processes, into the finest bloodvessels and capillaries. Aufrecht has done quite some experimental work in support of his contention.

It is thus clear that the aërogenous hypothesis of the origin of phthisis is explained by either a *hematogenous* or *lymphogenous* localization of the bacilli in the lungs. The frequency of tuberculosis of the glands, serous surfaces, and meninges speaks in favor of such origin of lung disease. The recent discoveries to the effect that a bacteremia is very frequent in phthisis support this contention.

Infection by Ingestion.—The most important mode of hematogenous infection in phthisis should be the ingestion of tubercle bacilli, although it by no means excludes the air passages as portals of entry, because germs inhaled through the mouth, nose, and throat may be swallowed and pass into the blood through the mucous membranes at any point of the gastro-intestinal tract. However, in the vast majority of cases, it would be with food, especially with milk from tuberculous cows, that the bacilli would enter the body and cause disease.

Simple as this theory appears, there are many objections to be considered before accepting it. The assertions of some authors that tubercle bacilli are invariably killed by the gastro-intestinal juices has been found largely incorrect, as was pointed out by Römer. To be sure, the gastro-intestinal juices may, and usually do, interfere with their rapid proliferation, and so may any fermentation in the intestinal tract, while the peristaltic movements of the intestines may soon remove them from the body; but they are not necessarily killed. Moreover, while a healthy, unbroken mucous membrane of the digestive tract is impermeable to tubercle bacilli, it is clear that a perfectly normal mucous membrane is very rare, considering the different kinds of food and its débris which pass through it, and the least disturbance in its anatomical structure or function may be sufficient to permit the passage of bacteria through its walls.

Experimental investigations have shown that feeding guinea-pigs, rabbits, and monkeys with tuberculous sputum, or with pure cultures of tubercle bacilli, is effective in infecting the animal. Moreover, it has been found that the bacilli may pass through the intestinal walls into the blood or lymphatics without leaving any trace on the walls of the canal.

Ravenel[1] conducted feeding experiments at the State Live Stock Sanitary Board of Pennsylvania and frequently observed extensive tuberculosis of the lungs and thoracic glands in animals which showed slight, or even no involvement of the intestine. He introduced into the stomach of a number of dogs tubercle bacilli suspended in an emulsion of melted butter and warm water, using a tube in order to prevent possible infection through the trachea. The dogs were killed after

[1] Jour. Am. Med. Assn., 1916, **46**, 613.

three and one-half to four hours, during active digestion, as much chyle as possible was collected, and the mesenteric glands were removed. Guinea-pigs were inoculated with this material. Tubercle bacilli were demonstrated in 8 of 10 experiments. The dogs were kept on soft food for some days before the experiment, and were purged with castor oil, in order to rid the intestine of all foreign matter which might injure the mucous membrane. Numerous sections of the intestine were examined, but no injury could be detected.

Because of this possibility of the tubercle bacilli entering the blood or lymph stream from the digestive tract, various authors have suggested the different parts of the canal, from the mouth to the rectum, as portals of entry of the bacilli, which are taken up by the blood and carried to the lungs where they finally stay and cause phthisis. Some have stated that irritated gums during dentition of infants offer a good portal of entry for the bacilli; the frequency of enlarged cervical glands at that period of life was cited as a good proof of the theory. Others have accused the tonsils, especially the pharyngeal tonsil. From the regional cervical glands some authors have traced the bacilli to the bronchial glands and finally to the lungs, though this has been shown by Wood[1] and Beitzke[2] not feasible for anatomical reasons. However, it must be acknowledged that even if there is no anatomical connection favoring the migration of bacilli from the cervical glands to the lungs, the microörganisms may be carried to any place by the blood. On the other hand, it must be mentioned that the tracheobronchial glands may be infected directly from the lungs by bacilli which have reached them with the inspired air.

The most conclusive proof of the tubercle bacilli entering the lungs *via* the digestive tract has been brought forward by Calmette and his school, also by Whitla, and many others. Calmette[3] denies dust containing tubercle bacilli as a strong factor in phthisiogenesis. He could not produce anthracosis in animals after subjecting them to prolonged inhalation of air saturated with lamp-black. Introducing dry, or moist, tubercle bacilli directly into the trachea by inhalation or insufflation, or even by inoculation, they were never found to reach farther than the bifurcation of the trachea. Introducing lamp-black into the stomach through a tube, thus excluding inhalation, or mixing it with food, anthracosis was soon produced in the lungs of the animals. Similarly, tubercle bacilli introduced carefully into the stomach through a tube with a view of preventing aspiration into the trachea, invariably produced tuberculosis.

Sir William Whitla's[4] experiments along these lines are very instruc-

[1] Ann. Rep. Henry Phipps Inst., 1906, **4**, 163.

[2] Virchow's Archiv, 1906, **184**, 1.

[3] Ann. de l'Inst. Pasteur, 1905, **19**, 601; 1906, **20**, 353. A more recent summary of Calmette's work and views is found in his L'infection bacillaire et la tuberculose, Paris, 1920. See especially pp. 145–156.

[4] Lancet, 1908, **2**, 135.

tive. He injected a mixture of China ink and water into the large vein in the ear of a rabbit. The animal was killed an hour later, and its lungs were found highly charged with carbon particles. He fed for four days a guinea-pig with an emulsion made by rubbing up finely powdered China ink in olive oil and water. The lung was found blackened by disseminated particles of carbon in the upper, and along the margins of the lower, lobes within from eight to twenty-four hours after a single dose. Whitla thus explains the migration of the carbon from the gastro-intestinal tract to the lungs: The carbon particles effect an easy entrance through the intestinal epithelial surface; reaching the lacteal or lymphatic paths they pass through the lymphatic glands of the mesentery, and finally, either inclosed in phagocytes or free, find their way into the thoracic duct to be poured into the venous circulation before being arrested in the capillaries of the lungs. Vassteenburgh and Grysez's experiments have also shown that it is easy to render an adult guinea-pig perfectly anthracotic without subjecting it to repeated inhalations of carbon particles. Considerable work along these lines has been done in this country. Schroeder and Cotton[1] found that no matter in what part of the body tubercle bacilli are inoculated, pulmonary disease may result.

Calmette's and Whitla's experiments have been repeated by many other authors but their results did not confirm these investigators. Thus, Cobbett[2] fed animals with Indian or Chinese ink, or with soot, using very much larger quantities than Calmette and Whitla used, and not once only, but many times, and in some cases daily for one or more weeks. In some cases it appears that he found in the older animals some amount of pigmentation of the lungs. But he was careful to examine a large number of control animals (a precaution which seems to have been omitted by Calmette and the others) and he found just as much pigmentation in them as in those animals which had been fed with carbon. In young animals pigment was not seen, whether they had been fed with carbon or not. It was clear that some amount of pigmentation of lungs was to be reckoned with in the older, town-bred animals, and Cobbett remembered that he was accustomed to see a considerable amount of carbonization in the lungs of adult guinea-pigs when he was working in Sheffield. He therefore decided to repeat the experiments with country-bred animals; and when this was done no pulmonary pigmentation was seen in any of the animals, whether they had been made to swallow the ink or not. The anthracosis was thus not necessarily due to the carbon introduced experimentally.

From these and many other experiments, contradictory though they are, we are safe in concluding that *tuberculous infection, including phthisis, may be acquired through the ingestion of tubercle bacilli, and that the digestive tract permits the passage of the bacilli, which are carried by the blood and lymph streams to the various points of least resistance,*

[1] Report of Bureau of Animal Industry, 1906, **23**, 31.
[2] Jour. Pathol. and Bacter., 1910, **14**, 563: The Causes of Tuberculosis, p. 146.

of which, in the human being, the pulmonary apices appear to be the most vulnerable.

It is, however, a question whether this mode of infection is the most common in spontaneous tuberculosis in humans. We must not overlook the fact, which has been established experimentally, that large numbers of bacilli are necessary to accomplish results; the normal gastro-intestinal tract can easily dispose of small doses of tubercle bacilli (see page 54.)

Ingestion of tubercle bacilli may result in tuberculosis of the cervical or mesenteric glands, depending on the point at which the bacilli enter the upper or lower parts of the digestive canal. From these glands the bacilli are taken up by the circulating blood and carried to the tracheobronchial or mesenteric glands, and to the lungs. In many cases the bacilli remain dormant in these glands indefinitely, causing no disease at all; in others, the latency lasts only for some time, when finally, because of some exciting cause, they flare up again, migrate with the blood stream and, localizing in the lung, cause phthisis, and we then think that we are dealing with a new infection.

Autopsies made by Gaffky,[1] Ungermann, Wollstein and Bartlett,[2] Ghon,[3] Hamburger,[4] and others have shown that in children both glandular systems—the abdominal and the thoracic—are affected in nearly the same proportion. Primary infection of the intestine is very rare in adults, though in children it is quite common. Behring, however, believes that all infections date back to early infancy when the bacilli are ingested, remain latent to flare up again in later years, causing disease of the lungs (see Chapter V). Of course, while making autopsies on adults who died from chronic tuberculosis it is difficult or impossible to find the point of primary inoculation. But in infants and children this may be done in many cases. Perhaps one of the best criteria is that in primary intestinal infection the mesenteric glands are implicated, while the intestinal mucous membrane may remain intact, and in secondary intestinal tuberculosis—the ulcerations so frequently found in phthisical subjects—the mesenteric glands are only rarely affected. Statistics of primary tuberculosis of the intestine in children are not in accord. From the data published by Orth, Eden, Councilman, Mallory and Pearce, Lubarsch, Wollstein and Bartlett, and many others, it appears that the percentage ranges from five to fifty. In children, a large proportion of these infections are due to bovine bacilli, as was already shown.

Milk Infections.—The type of bacillus discovered in a case of tuberculosis gives no positive clue as to the mode of its entry into the body. Of course, cow's milk contains tubercle bacilli more often than has been generally appreciated; but they are not always of the bovine type. The studies of E. C. Schroeder,[5] John F. Anderson,[6] Ravenel, and

[1] Tuberkulosis, 1907, **6**, 437.
[2] Am. Jour. Child. Dis., 1914, **8**, 362.
[3] Der primäre Lungenherd bei der Tuberkulose der Kinder, Berlin, 1912.
[4] Die Tuberkulose des Kindesalter, Vienna, 1912.
[5] Bull. No. 99, Bureau of Animal Industry, Washington, 1907.
[6] Jour. Infect. Dis., 1908, **5**, 107.

others, have shown this to be a fact in this country. In New York City, Alfred F. Hess[1] found virulent tubercle bacilli in 16 per cent of 107 specimens of milk retailed from cans. Inoculation experiments were carefully done and he found that guinea-pigs were infected with the milk, the cream, as well as the sediment. What is more noteworthy is that "commercially pasteurized" milk was also found to harbor tubercle bacilli. All bacilli were of the bovine type, with one exception, in which the human variety was discovered. M. Rosenau[2] compiled data concerning 551 samples of milk examined in which tubercle bacilli were found in 46, or 8.3 per cent, and he says that this may be taken as the average percentage for the entire country.

It appears, however, that the dangers of milk infections have been overestimated by many writers. This is shown by two sets of facts. In countries in which the population does not use cow's milk as a food at all, pulmonary tuberculosis is not at all lacking. This is the case in Japan, China, India, Egypt, etc., as has been shown by Kitasato.[3] In the federated Malay states[4] tuberculosis has recently been increasing considerably, though bovine tuberculosis does not exist at all; among more than 250,000 pigs killed in the Ipoh abattoirs during four years no case of tuberculosis was found. In China cattle have been found free from tuberculosis. On the other hand, even if infection with bovine tubercle bacilli takes place, as it does very frequently, the potential dangers are slight, even in children. Thus, according to Kossel,[5] during the period 1905 to 1909 the Imperial Health Department in Germany investigated 113 cases of tuberculosis of the udder in cows. It was found that in only 44 cases their milk was being consumed after being boiled. Altogether 360 persons consumed the milk from these cows in a raw state (151 children, 200 adults, and 9 whose age was not ascertained). It is noteworthy that they all, but 2, remained healthy. The 2 infected were infants, aged twenty-two and fifteen months. In both infants swelling of the cervical lymphatic glands occurred, and bacteriological examination, culturing, guinea-pig inoculation, and also cattle inoculation, revealed the presence of tubercle bacilli in these glands. But the general health of these infants remained good, no symptoms of any general disease appeared. They were reëxamined one and a half, and two and a half years later and found thriving. In both these cases the tuberculous milk was consumed for relatively long periods of time, one year, and eighteen months respectively. No other cases of tuberculosis were observed in that family. Hess,[6] in New York City, followed for three years 18 children who drank milk in which tubercle bacilli were demonstrated and found that all but 1 remained free from tuberculous disease. Only in 1 had tuberculous adenitis developed, and bacilli of the bovine type were cultivated from the pus of the gland abscess.

[1] Jour. Am. Med. Assn., 1909, **52**, 1011.
[2] Preventive Medicine, New York, 1913, p. 513.
[3] Sixth Intern. Congr. on Tuberculosis, 1908, **6**, 1.
[4] Jour. Am. Med. Assn., 1921, **66**, 785. [5] Deut. med. Wchnschr., 1910, **36**, 349.
[6] Jour. Am. Med. Assn., 1911, **56**, 1322.

Behring's idea that most tuberculous disease in adults has been acquired during infancy by ingestion of milk derived from tuberculous cows is thus not entirely sustained. Statistics gathered by various investigators have shown that the disease is not more frequently found in adults who during infancy were hand-fed than in those who were breast-fed.

Immunization by Bovine Tubercle Bacilli.—From the data arrayed in the preceding pages it appears that the tuberculous disease in children caused by bovine bacilli consists almost invariably in diseased glands, bones, joints, intestines, and skin, while fatal pulmonary infections are extremely rare. There is ample evidence to the effect that the adult human is practically immune to the bovine type of tubercle bacilli, even if his immunity to the human type of bacilli has not yet been established to the satisfaction of all. Younger individuals, when infected with the bovine type of the bacilli, find it more or less easy to cope with the situation and recover, even though they finally emerge with disfigurement, and perhaps crippled. But if the problem of tuberculosis was only that part which is produced by the bovine bacilli, it would not have by far the significance it has at present. In fact, several authors are of the opinion that these mild bovine infections immunize the organism against infections with the more virulent human type. In his investigations into the epidemiology of phthisis in Great Britain and Ireland, John Brownlee[1] arrives at the conclusion that "young adult" phthisis is less common in those counties from which a large amount of milk infected with tubercle is sent to the city of London. "Drinking tuberculous milk probably infects many persons with tuberculosis of whom the great majority recover. The American physicians making postmortems in France on persons dying of wounds, found that 75 per cent had evidence of healed, or caseating, tubercle in the abdominal glands, though infection of the thoracic glands was comparatively rare. Apparently the only reasonable assumption to account for this relation is that some degree of immunity may be developed during childhood which persists into early adult ages. The larger the number of infants, therefore, exposed to infection in childhood, the smaller the number found to take phthisis at the early adult age."

Distinction between Experimental and Epidemiological Infection.—From the data arrayed in this chapter it is clear that the enormous amount of experimental work done with the aim of shedding light on the problems of tuberculous infection has not entirely, or satisfactorily, explained the mode of entry of the bacilli into the human body. Of course, the contradictions in the experimental results obtained by various investigators may be explained easily by the various methods pursued in investigations, the different cultures and infecting doses employed, the character of the culture media, the species of animals used, etc. But after discounting all these variable factors, it remains

[1] An Investigation into the Epidemiology of Phthisis in Great Britain and Ireland, Medical Research Committee, Special Report Series, London, 1920, No. 46, p. 54.

a fact that *hardly any of the experimental modes of infection have completely imitated natural conditions as observed among human beings.*

It is therefore important to emphasize the immense distinction between experimental and epidemiological infection and immunity when considering tuberculosis in human beings. We know of many diseases which only rarely, or never, occur spontaneously in certain animals, but which may be induced experimentally. This is perhaps best seen in guinea-pigs which only rarely acquire tuberculosis under natural conditions, while experimentally they are found to be exceedingly susceptible, thus indicating that because we may induce a certain disease experimentally, this is not necessarily the manner in which the disease is engendered under natural conditions. It also shows that while we may infect a guinea-pig in a certain manner, it does not follow that humans are, under natural conditions, infected in the same manner.

We have seen that experimental infection does not produce the same results in different animals even when induced in the same identical manner, and with the same culture and dose of bacteria. When guinea-pigs are inoculated subcutaneously with bovine bacilli, the spleen is first affected, then the liver, while the kidneys are almost invariably spared; in the rabbit, next to the lungs, the kidneys are affected. The lung of the hen is practically refractory to the typus gallinaceus of the acid-fast bacilli. After inoculation of bovine bacilli the lymph glands are nearly always affected in the rabbit, but they are spared when human bacilli are used. Inasmuch as the visceral organs are affected after the virus is introduced through the subcutaneous route, it is evident that the bacilli pass through the regional lymphatics without producing visible harm. Comparative pathology abounds in examples which indicate that the various strains of the bacilli have different effects not only on different animals, but also on the same animal under different conditions. And when we bear in mind that the most common type of tuberculous disease, chronic phthisis, is distinctly a human disease which never occurs in animals spontaneously, and which has never been induced experimentally, it is apparent why experimental infection has not cleared up the problems of phthisiogenesis.

We have seen that *there is no agreement between authorities as to the most common channel of entry of the tubercle bacilli;* and Römer, perhaps the most indefatigable worker in the field of tuberculosis, arrived at the conclusion that *there is evidently some mode of transmission of this disease with which we are as yet unacquainted.*

It must, however, be mentioned here, a point which will be discussed in detail later on, that in human beings infection alone is not sufficient to produce disease; after all, disease occurs only in a comparatively small proportion of persons infected with tubercle bacilli. In other words, while there is no tuberculosis without tubercle bacilli, these microörganisms harm only those who are predisposed to the disease. We are more and more becoming convinced that phthisiogenesis is more a problem of predisposition than of bacterial infection.

CHAPTER III.

THE EPIDEMIOLOGY OF TUBERCULOSIS.

Ubiquity of the Tubercle Bacillus.—In our survey of the biological characteristics and the channels of entry of the tubercle bacilli we found that the virus of tuberculosis is ubiquitous; that it is found wherever civilized human beings congregate, because tuberculous human beings expectorate sputum containing these bacilli, and domestic animals affected with this disease are everywhere. It has been estimated that the number of bacilli discharged daily in the sputum of a single patient with advanced phthisis is as great as the number of human beings on the earth. The modest estimate mentioned by Cornet may be taken as near the truth—that 7,200,000,000 bacilli may be thrown off daily from a single patient. If we imagine each organism placed end to end in a single file, this number would constitute a chain not less than twelve miles in length.

Clinical and experimental medicine have shown conclusively that the expectoration of consumptives, milk from tuberculous animals, etc., are capable of causing infection; that these microörganisms may enter the body through wounds, as well as through the unbroken skin, and the mucous surfaces of the respiratory and alimentary tracts, etc. We have also shown that though there are many hindrances in the way of infection, still, when everything stated in the preceding chapter is considered, it is not surprising that one out of eight in civilized countries succumbs to the disease, but that the other seven escape its ravages.

Tuberculous Infection vs. Tuberculous Disease.—As a matter of fact very few escape infection with the tubercle bacilli, especially those living in large industrial cities. Making this statement it must be emphasized that *a distinction is to be made between tuberculous infection and tuberculous disease.* The latter refers to the disease known for centuries, ever since Hippocrates described it, as consumption, or the equivalent of the term found in all European languages. It is the disease which causes more than 95 per cent of the suffering, social and economic misery and deaths due to the tubercle bacilli. On the other hand, tuberculous infection covers all the cases in which the virus of tuberculosis has entered the body, irrespective of whether it has caused clinically appreciable disease, or not. *Tuberculous disease is always preceded by infection, but infection with the tubercle bacilli is not invariably followed by disease.*

Research of the past three decades has shown conclusively that

infection with tubercle bacilli is not invariably followed by that train of symptoms which we observe in phthisis; that it does not necessarily cause sickness, excepting an altered reactivity to tuberculin. Apparently more people harbor the bacilli within their bodies, or show traces of having harbored them, without knowing it at all, than such as suffer, or succumb, as a result of tuberculosis of the lungs, or of other organs. These persons are undoubtedly tuberculous, and there are many strong reasons for the assumption that, like other bacillus "carriers," they are liable to cause mild infections in others. But they are not at all phthisical in the clinical sense. Some of them are destined to become phthisical; in fact, practically all phthisis evolves from an infection acquired during childhood, as we shall show when discussing phthisiogenesis.

Frequency of Tuberculous Infection.—Careful and painstaking scientific investigations have shown that the frequency of tuberculous infection goes hand-in-hand with civilization, or contact of primitive peoples with civilized humanity. In modern large cities very few persons escape infection. Autopsies made with a view of ascertaining traces of tuberculous lesions, both active and healed, have shown that over 90 per cent of adults are thus affected among the civilized; but among primitive peoples, who have not come in contact with civilized conditions and humanity, no tuberculous changes are found at autopsies.

In Laennec's[1] classical work on diseases of the lungs published in 1831 we find the following in a footnote: "M. Lombard's investigations in the Children's Hospital at Paris show that of the children who die between one and two years of age, one-eighth are tuberculous; between two and three, two-sevenths; between three and four, four-sevenths; between four and five, three-fourths. In the succeeding years up to puberty, tubercles are found more frequently than before the fourth, but much less frequently than from the fourth to the fifth. Papavoine, of the same hospital, found that the number of tuberculous children between the fourth and eleventh years is greater than those who are not tuberculous, tubercles being particularly prevalent from the fourth to the seventh years. Their frequency is again increased about the twelfth and thirteenth years, and at fourteen and fifteen years the rate of prevalence is the same as at four and five. These results were obtained from investigations made on 910 children (388 boys and 522 girls); somewhat less than three-fifths were tuberculous."

Similarly, Henry Ancell[2] emphasized the extent of tuberculous disease in London as far back as 1840. In a paper on "Facts and Opinions Relating to Tuberculosis, with Commentaries," he cites the *Decennium Pathologicum* of Dr. L. K. Chambers, giving the results of the post-mortem examinations made in the mortuary of St. George's Hospital

[1] Traité de l'auscultation médiate et des maladies des poumons et du cœur, Paris, 1831, **2**, 125.

[2] Assn. Med. Jour., 1853, p. 1030; quoted from Karl Pearson, loc. cit., p. 19.

in the ten years, December 31, 1840, to December 31, 1850. The number of autopsies was 2046. The following are the figures:

	Birth to 15 years.	15 to 30.	30 to 45.	45 to 60.	Above 60.	All.
Total number of autopsies . . .	154	636	651	438	167	2046
Per cent of tubercle found . . .	29.7	35.8	25.8	19.6	7.7	26.1

It appears that these facts were entirely forgotten, and medical literature was silent about the extent of tuberculous infection, and changes in the bodies of many who have shown no indication of this specific disease during life, until in 1900 Naegeli[1] published his report of 500 autopsies at the Pathological Institute at Zurich. He found 71 per cent showed pathological changes due to tuberculosis. Among individuals under eighteen years of age, only 25 per cent showed such lesions, mostly of a grave character, often leading to a fatal termination. But in persons above eighteen years of age the proportion that showed traces of tuberculous infection reached 98 per cent. Of these, only 28 per cent died as a result of this disease, while the rest had tuberculous foci which were either altogether healed, or quiescent, or slowly progressing.

When first published this revelation appeared incredible, but then other pathologists investigated autopsy material along the same lines, and they practically confirmed Naegeli's findings. From the work of Harbitz, Scheel, Burckhardt, Lubarsch, Adami and McCrae,[2] and many others, it is clear that very few persons escape infection with tubercle bacilli before reaching the age of maturity. They have all found that *no matter what the cause of death may have been, whether the persons knew that they had been tuberculous or not, between* 50 *and* 100 *per cent of people living in large cities show active, quiescent or healed tuberculous lesions in some organs of their bodies.* On this point all are now in agreement, the only dispute which may be found in the literature consists in whether the percentage is only 70, or reaches as high as 100. Thus, Lubarsch[3] states that Naegeli exaggerated his findings, because of 7371 necropsies performed by Naegeli, Burckhardt, Risel and Lubarsch, only 4230, or 57.4 per cent, showed tuberculous changes; of 5796 necropsies on adults, 4017, or 69.2 per cent, showed such changes.

These autopsies showed another significant fact: *The newborn infant is invariably free from tuberculosis, indicating that infection, if it occurs at all, always takes place after birth.* Among infants dying during the first year of life from any cause, some are found presenting lesions of a tuberculous character, while beginning with the second year the number of infected children increases steadily, so that at the age of fifteen there are nearly as many tuberculous among them as among adults. In this country Martha Wollstein and F. H. Bartlett[4] reported 1320 autopsies performed at the Babies' Hospital in New York

[1] Virchow's Arch., 1900, **160**, 426.
[2] Tr. Sixth Internat. Congr. on Tuberculosis, 1908, **1**, 325.
[3] Virchow's Arch., 1913, **213**, 417.
[4] Am. Jour. Dis. Children, 1914, **8**, 364.

City on children under five years of age, of which 118, or 13.5 per cent, showed tuberculous changes. In Europe the proportion is even higher, as is evident from the findings of Naegeli, Burckhardt, Lubarsch, Hamburger, and many others.

In England autopsy material has shown the same conditions. Eastwood and F. Griffith, in London, and A. S. Griffith, in Cambridge,[1] have examined the organs and glands of 215 children who died from various causes in general hospitals, inoculating animals, etc. The proportion harboring tubercle bacilli is shown in the following table:

Age.	Number infected with tubercle bacilli.	Proportion infected.
0 to 2 years	6 out of 17	35 per cent
2 to 4 "	18 " 82	52 "
4 to 6 "	36 " 62	58 "
6 to 10 "	39 " 51	77 "
10 to 12 "	2 " 3	

Even conceding that among children who succumb the number of tuberculous is likely to be higher than among those who survive, the proportion is still very high—60 per cent of all children are shown to have been infected with tubercle.

Another series of autopsies on children has been reported by Harbitz.[2] In the Anatomical Institute at Christiania, Sweden, during 1898 to 1911, the bodies of 484 children who died from any cause were dissected. The ages ranged from birth to fifteen years. His results are given in the following figures:

Age.	Number examined.	Tuberculous lesions. Per cent.
0 to 1 year	201	20.0
1 to 2 years	65	26.2
3 to 4 "	44	31.8
5 to 6 "	28	67.9
7 to 10 "	53	62.2
11 to 14 "	53	81.1
15 "	40	80.0
Total	484	41.08

The anatomical picture was predominantly that of tuberculosis of the lungs and the lymphatic glands, especially those of the thorax. The younger the child, the more acute and progressive the lesion found. In only 1 case could he suspect congenital tuberculosis.

The most recent series of autopsies reported are those collected by A. Reinhart.[3] For eighteen months he made a special study of all cases that came to autopsy at the Berne Pathological Institute, looking for evidences as to the frequency of tuberculous lesions. In all he performed 460 autopsies. Among the 28 newborn infants no traces of tuberculosis were found; in 72 children under sixteen years of age, 29.16 per cent showed active tuberculous lesions, although only 16.8 per cent had succumbed to this disease. He again confirmed the results

[1] Report to the Local Government Board on Public Health, N. S., No. 88.
[2] Norsk mag. f. Laegevidesk., 1913, 5 R., 11, 1.
[3] Cor.-Bl., f. schweiz. Aerzte, 1917, 47, 1153.

of nearly all other pathologists to the effect that the number of tuberculous lesions increases with the advance of the age of the children. The infants under one year suffered the least, only 7.14 per cent. Among 360 cadavers of adults, 96.38 per cent were found with tuberculous lesions; negative results were encountered in only 13 cadavers, and of these 9 were under thirty years of age. Here again there is evidence that most tuberculous lesions heal: In 63.9 per cent of the adults the lesions were found healed; the older the individuals, the higher the proportion of healed lesions. It is also noteworthy that the difference between the incidence of healed lesions in town dwellers (92.9 per cent) and country dwellers (98.1 per cent) is rather slight.

Another point has been brought out by these autopsies which is of immense epidemiological and clinical importance. The tuberculous lesions found at the autopsies are not all active, nor were they the cause of death in many cases. Indeed, there were many which were latent, quiescent, or even healed. Thus among the 406 tuberculous bodies examined by Naegeli, 28.1 per cent had healed or latent lesions; among Burckhardt's 1452 autopsies he found 1221, or 84.1 per cent, tuberculous; but 39.4 per cent of them showed quiescent latent, or healed lesions, and Reinhart found 70 per cent inactive lesions. The results of nearly all other investigations show the same conditions.

Active and progressive lesions, leading to death, are characteristic of infancy; in fact, during the first year of life all lesions discovered at autopsies are those of generalized and progressive tuberculosis. Localized lesions are rare in childhood; they only make their appearance after the second year, and are still rare at ten years of age. Available pathological evidence tends to show that *the younger the individual infected with tuberculosis, the more likely he is to be killed by the disease, while the older the individual, the less is he likely to suffer from acute and progressive disease.* In fact, Lubarsch says that among older persons tuberculosis is a relatively harmless process, showing, as it does, a strong tendency to latency or healing. He illustrates this point by the following statistical facts:

Among 502 infants under one year examined after death, 4.58 per cent were found with tuberculous lesions, all of which were acute or subacute general tuberculosis, without any tendency to localization in a single organ. Of 123 children two years of age, 20.3 per cent were found with tuberculous lesions. All were also active and progressive, though there were already seen tendencies to localization of the process, but no calcification was noted. At three years of age 24.7 per cent of the bodies showed tuberculous changes, and in one some evidences of calcification were found microscopically in a tuberculous bronchial gland. He found that the number of active and fatal cases of tuberculosis keeps up at a high level until the age of fifteen, when localized tuberculosis begins to manifest itself, though the lesions still show tendencies to progression, and calcification is still exceptional. Thus, among 139 bodies of tuberculous individuals between one and sixteen years of age, only 33, or 23.7 per cent, showed

calcified foci, but none was completely healed—all were active and progressive in character.

Only after the seventeenth year of life are to be noted at autopsies latent and healed tuberculous lesions, and they keep on increasing in frequency, so that at the age of forty they are more frequent than progressive lesions. The following table, as well as Fig. 3, shows the point clearly:

Age.	Active lesions. Per cent.	Latent and healed lesions. Per cent.
17 to 20	77.4	22.6
20 to 30	76.7	23.3
30 to 40	52.6	47.4
40 to 50	38.9	61.1
50 to 60	33.5	66.5
60 to 70	23.3	76.7
70 to 80	14.7	85.3
80 to 90	9.3	90.7
90 to 100	0.0	100.0

These data must be considered underestimates, rather than overestimates, because while dissecting lungs and pleuræ slight and healed lesions may be overlooked, unless serial sections are made. Eugene L. Opie,[1] of St. Louis, attempted to overcome this possible source of error by an ingenious method. While making autopsies on 93 children, under eighteen years of age, and 50 adults, he roentgenographed each lung, and since calcium salts are impervious to the roentgen rays, small nodules which could not be detected on inspection and dissection were easily discovered. He thus found that partially calcified foci containing caseous material of soft, friable consistence are conspicuous in roentgen-ray plates. In some specimens tuberculous nodules seen on the plate could not easily be found on dissection, but careful search always revealed them. In all doubtful cases concerning the nature of a lesion, microscopic examination of the tissue was made. It is thus clear that Opie's work was very carefully done.

His results are given in the following table:

Age (years).	Number of autopsies.	Tuberculosis.				In those who have died with other diseases.
		Present.		Fatal.	Non-fatal	
		Number.	Per cent.			
Under 1	43	4	9.3	4	0	0.0
1 to 2	16	1	6.2	1	0	0.0
2 to 5	14	6	42.8	3	3	27.3
5 to 10	11	5	45.5	2	3	33.3
10 to 18	9	6	66.7	1	5	62.5
18 to 30	6	6	100.0	1	5	100.0
30 to 50	23	23	100.0	1	22	100.0
50 to 70	15	15	100.0	1	14	100.0
70 and over	6	6	100.0	0	6	100.0

[1] Jour. Exper. Med., 1917, **25**, 885; **26**, 263.

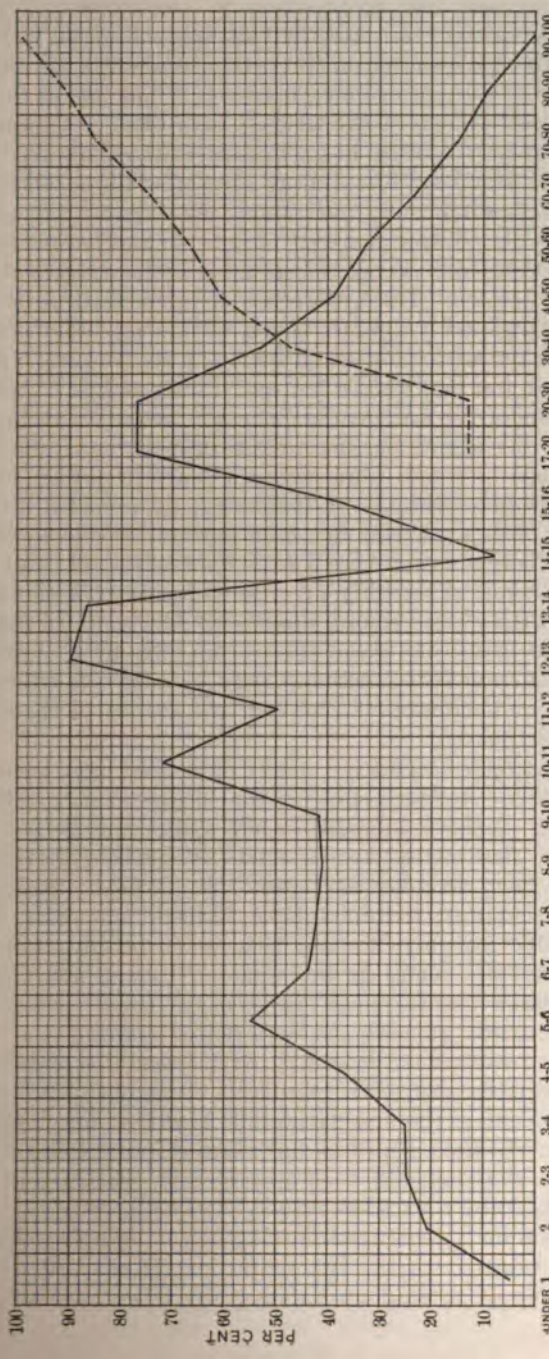


FIG. 3.—Distribution of tuberculous lesions at various age periods as found at autopsies performed by O. Lubarsch. Black line, active lesions; dotted line, latent, quiescent, and healed lesions.

Here again it is clear that in this country the number of persons infected with tuberculosis is not less than has been observed in Europe, though most of the lesions have not been the cause of death, but have healed, leaving but scars, or calcified nodules. "The age of incidence of focal tuberculous lesions of the lungs," says Opie, "demonstrates that they have their origin in most instances in childhood. Focal lesions which heal have been found at all ages after the second year of life, but in more than half of all individuals these lesions are acquired between the ages of ten and eighteen years. In the period between eighteen and thirty at least 85 per cent of all individuals have acquired focal tuberculous lesions. The occurrence of tuberculous infection in the lungs, in the regional lymphatic nodes, or in some other organs of the body, such as the gastro-intestinal tract and its lymphatic system, is nearly universal, but doubtless a few individuals escape. That focal tuberculous lesions of the lung are occasionally acquired during adult life is shown by the slight increase in the proportion of those with these lesions as age increases from eighteen years to old age."

The frightful tuberculization of humanity, as revealed by these autopsy findings, was explained by some authors as due to the fact that in hospitals there is a concentration of tuberculous sick, and among children who succumb at an early age, the percentage of tuberculous should be much higher than among those who survive until maturity. But it must be recalled that these autopsy findings were obtained in children who died from all causes, and that in many the tuberculous lesions were found incidentally, although the causes of death were entirely different diseases.

Reliability of Autopsy Statistics.—Many objections have been raised against these autopsy statistics showing that nearly every adult living in a modern city harbors tubercle bacilli within the body. Some have maintained that many non-tuberculous changes in the lungs and pleura have been included as "latent" or "healed" tuberculosis. But Naegeli, Burckhardt, Reinhart, Opie, Griffith, and many others, state distinctly that extreme care had been taken before pronouncing doubtful pathological changes as tuberculous. Some, like Opie and Reinhart, have made microscopical studies of the tissues before deciding; Griffith inoculated guinea-pigs, etc., before deciding.

It has also been suggested, especially by Cornet, that these latent lesions were caused by avirulent, or mildly virulent tubercle bacilli, perhaps even by some of the non-pathogenic acid-fast microörganisms which abound in nature. But this has been disproved for the first time by Loomis, who injected such glands into rabbits and found that they were infected with tuberculosis. Cobbett,[1] working for the Royal Commission on Tuberculosis in England, found that definitely caseous nodules taken from the lymphatic glands of children might be quite incapable of setting up tuberculosis when emulsified and

[1] The Causes of Tuberculosis, p. 70.

injected into animals, even when the injections were made in such a susceptible animal as the guinea-pig. This was surprising, but what was more surprising still, the caseous matter thus shown to be totally devoid of infective power might contain plenty of well-formed tubercle bacilli, easily visible under the microscope. Similar experiences were recorded by A. S. Griffith, Weber, and others. This would tend to confirm Cornet's view that the lesions were produced by avirulent, or mildly virulent, tubercle bacilli, and for this reason the disease they produced was not active, nor fatal. But other investigators did find virulent tubercle bacilli. Thus, Lydia Rabinowitsch[1] found that completely calcified glands, in which no tubercle bacilli could be discovered microscopically, were still capable of infecting animals. Eastwood and Griffith even cultivated tubercle bacilli from glands of 72 children, 34 of whom were apparently non-tuberculous.

It is thus clear that the tubercle bacilli found in the healed lesions of persons who have succumbed to diseases other than tuberculosis are often alive and virulent.

The objection has also been raised that the autopsy material obtained in morgues in large cities represents the lowest grades of society, the poorest strata of population, who are most likely to succumb to tuberculosis, while the well-to-do, or self-supporting elements of society, even in cities, are by no means tuberculous to such an appalling extent. But it is the poor who present the problem of tuberculosis most acutely. Moreover, Naegeli pointed out that his material was not exclusively of the lowest strata of society. Forty per cent at least were country folk, and 6.5 per cent were private patients; only in 22.5 per cent was tuberculosis the cause of death, as against 28 per cent occurring among the general population of the Canton of Zurich, thus showing that the persons on whom he made his autopsies were not excessively tuberculized.

Better confirmation of these findings was, however, supplied by several series of autopsies made on persons who had enjoyed good health but succumbed to accidents, or acute diseases. Among 826 autopsies made on such individuals, Birch-Hirschfeld[2] found 171, or 20.7 per cent, with tuberculous lesions. Of these, 105, or 12.7 per cent, were healed lesions; 31, or 3.8 per cent, were actively advanced; 35, or 4.2 per cent, were latent or mildly active. Similar results were reported by J. G. Mönckenberg,[3] who made autopsies on 85 soldiers fallen in the World War. In 25, or 31.76 per cent, he found distinct evidences of active, latent, or healed tuberculosis. In 5 cases the lesions were so active that they may have been the cause of death, but in the remaining 22 cases the tuberculous lesions were incidental findings. Likewise, Hart[4] made autopsies on 573 soldiers

[1] Berl. klin. Wchnschr., 1907, p. 35.
[2] Deutsch. Arch. f. klin. Med., 1899, **64**, 58.
[3] Ztschr. f. Tuberk., 1915, **24**, 33.
[4] Ibid., 1919, **31**, 129.

who died from non-tuberculous diseases during the war, and found that 196, or 34 per cent, had tuberculous lesions, of which 151, or 26.8 per cent, were "obsolete."

Extent of Tuberculous Infection among the Living.—The extent of tuberculous infection among the living population has been ascertained by the application of the tuberculin test, which is even more delicate than the macroscopic examination of the body after death, showing, as it does, the number of persons infected with tubercle bacilli and who have survived, or have not at all suffered as a result of the infection. No matter how slight the lesion produced by the tubercle bacilli, the tuberculin test reveals it.

Extensive investigations have been made along these lines, and it was found that there are very few adults living in cities who do not react to tuberculin. Those who live in tubercle-laden surroundings hardly ever escape infection. Pollak[1] found that in Vienna 96 per cent of children of tuberculous parentage were infected before they reached the fourth year of life. In Spain, Munoyerro and Frias[2] found tuberculosis in 195 of 3000 children from one to three years of age at a Madrid asylum. The tuberculin tests showed 6.3 per cent of infections at this early age, and the tuberculous cadavers formed 9.3 per cent of the total. In Argentina, Garrahan[3] applied the tuberculin test, often three or four times, to 1214 children at Buenos Aires, between two and sixteen years of age. He found that of the children between fourteen and sixteen, 75 per cent showed signs of tuberculous infection. Mantoux[4] found that 84 per cent of the children in Paris were infected before they reached the fifteenth year. A more recent investigation by Germaine Mioche,[5] who applied the tuberculin test to 2784 children at Paris, gave the following proportion of positive reactions:

Age.	Number of cases.	Per cent positive.
Under 3 months	298	3.7
3 to 6 months	459	7.2
6 to 12 months	583	16.8
Under 1 year	1340	10.6
1 to 2 years	247	24.3
2 to 5 years	467	56.8
5 to 10 years	525	67.4
10 to 15 years	302	82.7

In New York City the author[6] has found that children living with their tuberculous parents are infected to the extent of 84 per cent at the age of fourteen, as can be seen from the table and the attached diagram (Fig. 4). Similar results have been obtained while testing large numbers of children of tuberculous parentage in various European cities.

[1] Brauer's Beitr., 1911, **19**, 469.
[2] Arch. Espanoles de Pediatria, 1919, **3**, 532.
[3] Semana Medica, 1919, **26**, 771.
[4] Sémaine méd., 1909, **29**, 371; Presse méd., 1910, **18**, 10.
[5] Le Nourisson, 1920, **8**, 41.
[6] Arch. Pediat., 1914, **31**, 96, 197.

Taking apparently healthy children at random, *i. e.*, those who do not live in homes harboring evidently tuberculous persons, it appears that they are also infected in large numbers. Hamburger[1] found that at the age of fourteen 94 per cent of the children of artisans in Vienna show signs of infection with tuberculosis. Calmette[2] at

TABLE SHOWING EXTENT OF TUBERCULOUS INFECTION AMONG THE POORER CLASSES IN NEW YORK CITY BASED ON THE APPLICATION OF THE TUBERCULIN TEST ON 1280 CHILDREN UNDER FIFTEEN YEARS OF AGE.

Age.	Percentage giving positive reactions among Children of tuberculous parents. Number of cases.	Per cent.	Children of non-tuberculous parents. Number of cases.	Per cent.
Under 1 year	33	15.15	56	10.07
1 to 2 years	49	55.10	39	33.33
3 to 4 "	90	68.88	80	41.25
5 to 6 "	95	65.26	106	50.00
7 to 10 "	244	71.31	173	64.74
11 to 14 "	181	74.58	134	69.40
14 "	37	83.79	20	75.00

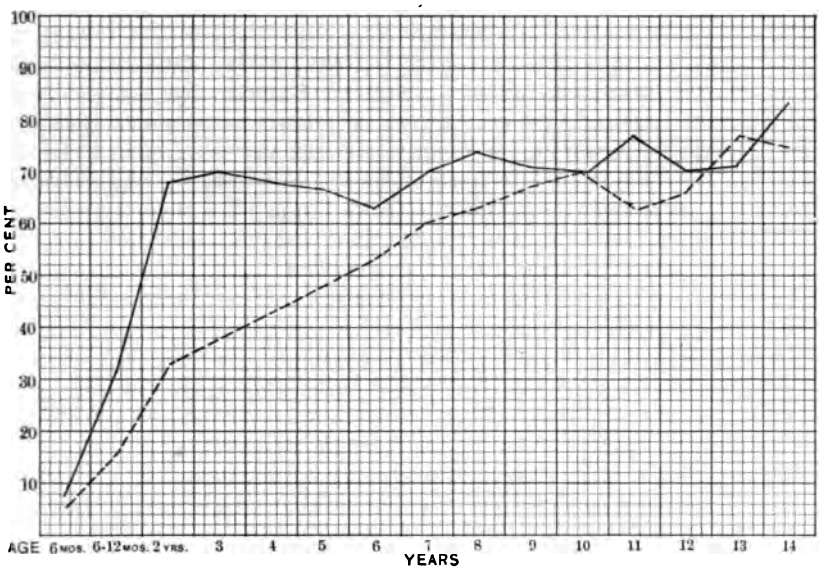


FIG. 4.—Proportion of children reacting to the cutaneous tuberculin test. Black line represents 692 children of tuberculous parentage in New York City; dotted line represents 588 children of non-tuberculous parentage in New York City.

Lille, France, testing 1226 persons of all ages taken at random from diverse social strata, all apparently healthy, found that during the first year of life only 9 per cent were infected, but the percentage kept on increasing, so that at the age of fifteen and over, 87 per cent

[1] Die Tuberkulose im Kindesalter, Berlin, 1913.
[2] Grysez et Letulle, Presse méd., 1911, **19**, 651.

were infected. In New York City the author[1] found while testing children of poor, but non-tuberculous parentage, that under one year of age 10 per cent were infected; between one and two years of age, 33.33 per cent, and the proportion giving positive reactions to tuberculin kept on growing steadily with advancing age, so that at the age of fourteen, 75 per cent of "reactors" were found.

It is well known that the von Pirquet test, which was used in these cases, is occasionally negative when applied the first time, but is positive when applied a second or third time. For this reason some who have applied the test but once found a lesser number of reactors. J. B. Manning and H. J. Knott,[2] in Seattle, tested 228 children, aged ten to fourteen years, coming to the Children's Tuberculosis Clinic, the large majority of whom were from tuberculous homes. Of 166 with a definite history of exposure, 84, or 50.6 per cent, gave a positive von Pirquet test, though 82.1 per cent of these children showed no clinical evidences of tuberculosis. Of 62 children with no history of exposure, 14, or 22.8 per cent, were reactors. But they used only one-half strength of tuberculin, and when found negative after the first application the test was not repeated. Had they applied it twice or three times, and in full strength, the proportion of reactors would undoubtedly have been higher. George H. Cattermole[3] tested children in Boulder, Colorado, where there is no overcrowding, but plenty of good food and sunshine. Probably one-half the families in Colorado contain one or more adult consumptives. It would be expected that the number of reactors should be quite large. Yet only 38 per cent were found to have been infected. This anomaly may be explained by the superior social and economic conditions, but it seems to me that the following reasons are more plausible: The number of children was rather small, only 66; if he had extended his investigations the results might have been different; he applied the test but once in most cases, using the von Pirquet and the Moro tests. At any rate it appears that opportunities for infection were not altogether counterbalanced by superior climatic and economic conditions.

While it is in large industrial cities that tuberculosis is most widespread, as is shown by the high morbidity and mortality from the disease, infection is not lacking in rural communities of civilized countries. Investigations made by Jacob,[4] Hillenberg,[5] Overland,[6] and others have shown that in villages, where a case of open tuberculosis had not been seen for many years, the people living under good economic and hygienic surroundings, and where the milk supply was practically free from tuberculous contamination, 25 per cent of the

[1] Arch. Pediat., 1915, **32**, 20.
[2] Am. Jour. Dis. of Children, 1915, **10**, 354.
[3] Jour. Am. Med. Assn., 1915, **65**, 782.
[4] Die Tuberkulose und die hygienische Misstände auf dem Lande, Berlin, 1911.
[5] Tuberkulosis, 1911, **10**, 254.
[6] Internat. Zentralbl. f. Tuberkulose, 1914, **8**, 635.

school children and about 45 per cent of the adults gave positive reactions to tuberculin, indicating that they had not escaped tuberculous infection. Here we find that the effect of infection is only an altered reactivity to tuberculin, and not phthisis. The reasons for this phenomenon will be discussed later on.

Tuberculosis among Primitive Peoples and Races.—*The only regions free from tuberculosis appear to be those inhabited by primitive peoples who have not come in contact with civilization.* Thus, the American Indian, before the advent of the white man on this continent, knew nothing of the disease, as was shown by Woods Hutchinson,[1] Hrdlicka,[2] and others. Nor do the savage and barbarian races of Central Africa and Asia seem to have had experience with tuberculosis, until the whites brought it to them. Among these primitive peoples the tuberculin reaction is always negative, and autopsies made on their dead reveal no active or healed tuberculous lesions, as is the case with newborn infants among Europeans. But it appears that as soon as these peoples come into contact with civilized man they are infected in large numbers. This was observed among the American Indians, the native tribes of Australia and Africa, etc. The application of the tuberculin test among these races by Calmette,[3] Metchnikoff,[4] Ziemann,[5] and others has shown clearly that the frequency of tuberculous infection depends directly on their contact with civilization. It is altogether absent, or extremely rare, among those races who have recently met the white man, but the proportion grows in direct ratio to the intensity of immigration of European settlers, and with commercial interchange between them and civilized humanity. It is also evident that their immunity from this disease before the advent of the white man was not due to racial or climatic conditions, as was suggested by some earlier writers, but solely to the absence of tubercle bacilli, because as soon as these are imported, the natives display a striking vulnerability to the disease, which is greater the longer they have been protected against the importation of tubercle bacilli.

Racial Differences in Susceptibility to Tuberculous Infection.—A study of the epidemiology of tuberculosis also teaches that the dangers of tuberculous infection depend on the length of time a people has been exposed to the disease. Thus, when primitive peoples who has never been affected with this disease come into tubercle-laden surroundings, they are soon infected and the disease runs an acute and fatal course in nearly all cases. This is often the case with savages and barbarians brought to Europe or America: They almost invariably acquire tuberculosis, and succumb in a short time. The American Indians, coming in contact with the whites, and incidentally with the

[1] New York Med. Jour., 1907, **86**, 624.
[2] Tuberculosis among Certain Indian Tribes of the United States, Washington, 1909
[3] Ann. de l'Instit. Pasteur, 1912, **26**, 497.
[4] Ibid., 1911, **25**, 785.
[5] Centralbl. f. Bakteriol., 1913, **70**, 118.

tubercle bacillus, are being decimated by the disease which runs an acute and fatal course among them, and the same is true of the negro population in this country.

A drastic illustration has been reported by Cummins[1] from Egypt, where the Sudanese soldier, recruited from tribes among which tuberculosis is practically unknown, is much more liable to tuberculosis than the Egyptian soldier who has been raised in a region where the disease has been quite common for centuries. In former times slaves of the Sudanese race were the cheapest in the market, because it was assumed that a large number would contract the disease and die.

FIG. 5.—Mortality from pulmonary tuberculosis in the United States Registration Area according to race. (Hoffman.)

During the recent World War this point was clearly brought out when African and Asiatic troops were transported to Europe in large numbers. S. Lyle Cummins[2] reports that there were more deaths from tuberculosis in the British armies in France during 1917–1918 in a few companies of Africans than among the whole of the British troops in France. The contrast between the case mortality was equally striking: 5.7 per cent among the British and 56 per cent among the Africans. These African labor units were subjected to careful medical examination before leaving their country, which shows that they had acquired tuberculosis in France. The Fijian labor units had to be repatriated on account of the prevalence of tuberculosis among them. The Indian divisions in France during 1916 showed a tuberculosis incidence of 27.4 per 1000, as compared with a case incidence of 1.1 per 1000 among British troops. Similar conditions were observed among the Indian and Chinese labor contingents. A. Borel[3] reports the same among the French African troops. He found that among those who recently arrived from Senegal only 4 or 5 per cent gave positive cutaneous reactions, indicating that they were hardly tuberculized. But after staying in France for some time the death rate from tuberculosis increased to 11.14 per cent. On the other hand, American negroes, who had been exposed to tuberculous infection in the United States, did not show an excessive tuberculosis mortality while serving in France with the American Expeditionary Forces.

[1] Tr. Soc. Trop. Med. and Hyg., 1911–1912, **5**, 245.
[2] Internat. Jour. Public Health, 1920, **1**, 137.
[3] Ann. Inst. Pasteur, 1920, **34**, 105.

This is exemplified again by conditions observed among the immigrants to the United States. The Irish and Sicilian immigrants, and to a lesser extent the Hungarians, Slavonians, and Scandinavians, mostly hail from agricultural parts of their native countries where they have known very little of tuberculosis. In this country, working in closed factories, and coming in contact with tuberculous fellow-workmen, many soon contract the disease, which runs an acute course, terminating fatally in a large proportion of cases. Among immigrants coming from countries or cities where they have been exposed to infection for generations, as is the case with the English, Germans, and especially the Jews, the rates of tuberculous mortality are much lower.

When speaking of race influence on the incidence and mortality from tuberculosis, the facts just mentioned must always be borne in mind. Tuberculosis appears not to be a racial problem—there are no races which are more or less vulnerable to the disease, because of their ethnic peculiarities, such as height of the body, color of the skin, eyes and hair, or other somatic or morphological traits which distinguish one race from another. One human race, or ethnic group, when first meeting with tubercle bacilli, is as vulnerable as another. It is only after they have been exposed for many generations to the disease that they acquire a certain resistance against infection, which, though occurring in almost everyone who has been exposed to infection, is less liable to cause disease than in races which present virgin soil to the bacilli. The mechanics of this acquired immunity will be discussed later on.

MORTALITY FROM PULMONARY TUBERCULOSIS PER 100,000 POPULATION.

Country.	1861 to 1865.	1866 to 1870.	1871 to 1875.	1876 to 1880.	1881 to 1885.	1886 to 1890.	1891 to 1895.	1896 to 1900.	1901 to 1905.	1906 to 1910.
United States	...	...	...	...	...	...	...	...	171	147
England and Wales	253	245	222	204	183	164	146	132	122	111
Scotland	252	262	248	229	211	189	174	165	145	
Ireland	..	183	191	200	208	212	214	213	215	191
Australia	..	..	..	..	122	121	107	94	89	75
New Zealand	..	..	..	..	91	84	81	78	70	62
*Ontario Province	..	..	..	..	125	116	114	141	129	113
Germany	..	..	..	361	348	314	224	194	186	159†
Prussia	..	..	..	317	312	290	247	208	191	162
Bavaria	..	..	..	..	..	..	287	262	243	214†
Saxony	..	..	251	251	244	236	212	194	154	135†
Baden	..	..	..	..	312	297	278	244	217	183
*Austria	..	..	..	377	393	383	394	345	340	305
Switzerland	..	..	..	200	209	213	199	190	189	176†
Netherlands	...	...	...	...	...	..	189	165	133	125
*Belgium	305	305	335	323	301	..	165	142	118	102†
France	..	..	..	..	..	..	255	249	265	277†
Italy	..	..	..	..	..	137	100	106	116	123†
Spain	..	..	..	..	..	..	..	..	148	135
Denmark	..	..	..	262	249	231	200	160	149	134†
Norway	..	..	108	126	140	144	173	206	196	200†
Finland	..	374	414	367	255	256	261	273	291	
Serbia	..	..	..	..	..	..	251	231	280	297†
*Hungary	..	..	..	..	..	..	..	364	397	374
Chile	..	..	..	..	..	235	269			
Japan	..	..	..	..	..	101	136	145	146	154†

NOTES.—All figures refer to pulmonary tuberculosis, except those marked * which include all forms of tuberculosis.

Figures in the last column marked † are only for 1906–1908.

Geographical Distribution.—Sixty years ago Hirsch, in his classical study of *Geographical and Historical Medicine*, arrived at the conclusion that tuberculosis is a disease of all times and all countries. With our present knowledge we have not discovered any proofs to the contrary. Observations in every part of the habitable globe show that the presence or absence of the disease is determined less by geographical location, or climatic phenomena, than by social and economic conditions and, above all, by the presence or absence of the tubercle bacillus. We have shown in the preceding pages that its absence in certain countries cannot be ascribed to either an immunity of the population, nor to the climate in which they live, nor to the altitude on which they have been located. Indeed, it is obvious that as soon as the tubercle bacilli are introduced among any people in any geographical location, the disease is not slow in making its appearance. The comparative absence of tuberculosis in the Rockies, the Andes, and other mountainous regions, in former times was apparently due to the scarcity of population, and the peculiarity of the occupations there pursued. In the mountainous regions of the United States tuberculosis was scarce before consumptives began to immigrate in

DEATH-RATES FROM PULMONARY TUBERCULOSIS PER 100,000 POPULATION IN VARIOUS CITIES.

City.	1881 to 1885.	1886 to 1890.	1891 to 1895.	1896 to 1900.	1901 to 1905.	1906 to 1910.
London	222	197	185	175	157	132
Edinburgh	212	191	180	187	157	114
Glasgow	311	250	227	195	170	140
Dublin	346	341	335	317	309	268
Belfast	382	402	382	329	307	235
Paris	441	440	409	379	390	374
Berlin	..	..	..	..	..	188
Hamburg	..	..	238	200	169	137
Munich	389	348	312	303	269	226
Dresden	376	334	283	247	224	180
Breslau	331	313	342	321	318	271
Amsterdam	238	234	204	185	144	138
Rotterdam	219	192	188	170	133	127
The Hague	199	179	163	160	128	124
Vienna	685	576	474	381	336	274
Prague	728	609	512	472	525	385*
Budapest	715	591	434	376	367	340
Trieste	522	491	439	402	396	369
Christiania	320	287	282	274	229	183*
Stockholm	344	303	269	246	227	230
Copenhagen	273	246	198	180	144	136
Petrograd	547	449	384	321	305	301
Moscow	411	393	391	324	268	258
Milan	335	307	284	204	232	220
Turin	240	222	250	234	225	183
Sydney	193	157	119	98	98	72
Melbourne	233	213	182	153	139	109
Montreal	282	256	235	250	197	163*
Toronto	203	207	242	234	174	
Rio de Janeiro	548	..	446	474	455	402

Figures marked * indicate that the death-rate in the last column is only for 1910.

search of health. Brown, investigating conditions in El Paso, Texas, found that the testimony of physicians is to the effect that deaths due to this disease are rare among the indigenous population; E. A. Sweet[1] finds this to be true of the entire southwest region of this country, and Cattermole confirmed it in Colorado. But it appears that the infection of people living under good sanitary, and above all, economic conditions does not always produce phthisis, especially in regions where outdoor life is the vogue.

Incidence among Rural and Urban Residents.—Of greater influence than climate and altitude appears to be life in the city, when compared with life in the country, as regards the morbidity and mortality from tuberculosis. It appears that country dwellers, while not exempt from infection with tubercle bacilli, are less likely to suffer from phthisis than city residents. Thus, the average death-rate from tuberculosis of the lungs in the registration area of the United States during the decade ending with 1909 was 154.7 per 100,000 population, but in the cities of the registration area the rate was 177.4 against

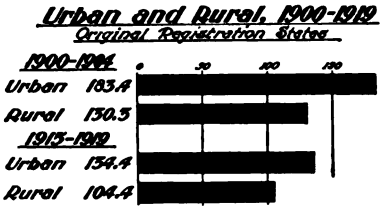


FIG. 6.—Mortality from pulmonary tuberculosis in the United States Registration Area among city and country dwellers. (Hoffman.)

a rural death-rate of but 124.1. These differences would be even greater if we included the rural centers in which factories, mills, mines, etc., are located and where the workers live to all intents and purposes under the same conditions as those in the cities. Such differences in the mortality from phthisis are found in every country where vital statistics are gathered. In England and Wales the mortality per million population was in 1913: London, 1335; England and Wales, 1004; rural districts, 742; all urban districts, 1075. The table on page 76 shows the high mortality-rates from this disease in large cities in various parts of the world. When compared with the rates for the entire country, as given on page 75, the differences are clear.

The establishment of sanatoriums for consumptives in rural districts during recent years has apparently increased the mortality from this disease in certain country districts. Thus, in 1910 the death-rates from pulmonary tuberculosis in the State of New York were: in cities, 165.7; in the rural districts, 120.1, while in Colorado, the Mecca of

[1] Public Health Reports, 1915, **30**, 1059, 1147, 1225.

American consumptives, the rates were: cities, 288.2; in rural districts, 155.9. It is thus evident that with superior climate and altitude, Colorado has a higher mortality from pulmonary tuberculosis than the State of New York. Of course, the reason is that most of the fatal phthisis in Colorado is imported. According to H. B. Whitney[1] the Coloradoans are practically immune to tuberculosis. He brings statistics showing that only a small percentage of deaths from this disease occur among the natives of Colorado.

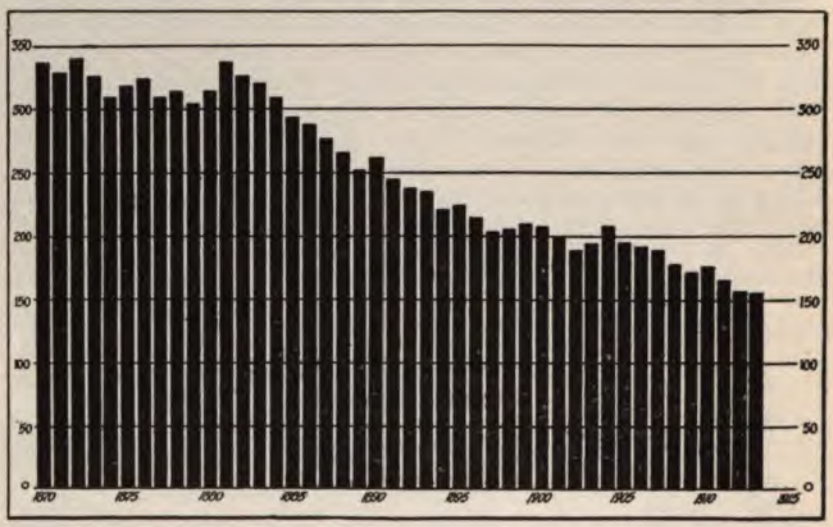


FIG. 7.—Decline in the mortality from pulmonary tuberculosis in American cities since 1870. (Hoffman.)

Wherever available, statistics show clearly that there is more fatal tuberculosis in cities than in the country. The reasons for this disparity should be sought not only in the outdoor life which country dwellers indulge in more than city people, but more in the difference in social and economic conditions.

The higher mortality from phthisis in towns as compared with rural districts appears to affect only the male population, as has recently been shown by Benjamin Moore.[2] In the country districts of England and Wales it appears that the mortality of females is higher than that in the cities. In both town and country nearly twice as many girls as boys die from phthisis between the ages of ten and fifteen. While until the twentieth year the mortality from pulmonary tuberculosis of both sexes is greater in rural districts than in urban districts, between the twentieth and thirtieth years the condition in the towns become reversed. After the thirtieth year the

[1] Colorado Medicine, 1919, **16**, 268.
[2] Lancet, 1918, **2**, 618.

disease preponderates greatly among urban males as compared with urban females. Moreover, the disparity of the phthisis mortality just mentioned is a recent phenomenon; it was not observed in the returns of seventy years ago. It is apparently due to the recent changes in the social and economic conditions of the population brought about by the recent conditions among the working classes.

MORTALITY FROM PULMONARY TUBERCULOSIS IN AMERICAN CITIES, 1900 TO 1919. RATES PER 100,000 POPULATION.

	1900–04	1905–09	1910–14	1915–19
Atlanta, Ga.	245.5	193.7	148.9	174.0
Baltimore, Md.	235.5	233.9	204.5	174.5
Birmingham, Ala.	.....	.....	227.4	183.8
Boston, Mass.	223.2	180.9	156.6	151.9
Bridgeport, Conn.	183.9	162.5	108.6	129.0
Buffalo, N. Y.	123.2	126.6	129.5	137.3
Cambridge, Mass.	207.6	224.9	191.4	186.0
Chicago, Ill.	157.8	163.7	146.8	130.3
Cleveland, Ohio	122.8	117.9	118.8	126.3
Columbus, Ohio	203.6	178.7	151.5	128.2
Dayton, Ohio	184.0	191.0	136.9	119.9
Denver, Col.	378.0	363.0	274.6	255.2
Detroit, Mich.	113.0	96.2	88.3	96.9
Fall River, Mass.	181.8	139.7	139.5	149.8
Grand Rapids, Mich.	105.4	82.8	77.7	74.1
Indianapolis, Ind.	192.8	184.7	167.1	149.2
Jersey City, N. J.	233.2	208.4	155.5	139.7
Los Angeles, Cal.	320.8	267.2	232.7	192.9
Louisville, Ky.	211.5	207.7	195.4	166.1
Lowell, Mass.	160.1	139.6	107.5	115.7
Memphis, Tenn.	229.8	226.2	242.1	231.5
Milwaukee, Wis.	128.7	117.3	98.3	82.5
Minneapolis, Minn.	114.9	106.0	122.2	119.0
Nashville, Tenn.	303.2	246.9	197.9	178.9
Newark, N. J.	239.4	222.8	173.5	149.2
New Haven, Conn.	177.6	164.3	118.8	99.0
New Orleans, La.	324.2	272.7	252.8	264.0
New York, N. Y.	221.7	202.0	181.0	161.5
Oakland, Cal.	162.7	158.5	112.6	108.6
Paterson, N. J.	188.8	163.7	141.6	128.2
Philadelphia, Pa.	214.9	207.8	178.0	168.8
Pittsburgh, Pa.	133.7	120.6	106.4	120.6
Portland, Ore.	107.7	86.9	77.9	72.7
Providence, R. I.	213.3	155.3	130.1	129.8
Richmond, Va.	266.4	224.8	200.5	172.7
Rochester, N. Y.	133.8	136.4	105.0	89.0
San Francisco, Cal.	279.5	191.8	169.0	164.3
Scranton, Pa.	96.1	81.6	86.4	78.4
Seattle, Wash.	99.0	77.7	86.9	65.5
Spokane, Wash.	117.2	94.0	80.1	62.0
St. Louis, Mo.	196.3	187.1	146.1	125.4
St. Paul, Minn.	108.8	106.6	115.0	113.5
Syracuse, N. Y.	142.3	117.8	97.3	80.2
Washington, D. C.	274.8	243.1	205.8	160.8
Worcester, Mass.	180.0	147.9	110.4	118.8
Average	198.9	181.5	158.9	145.1

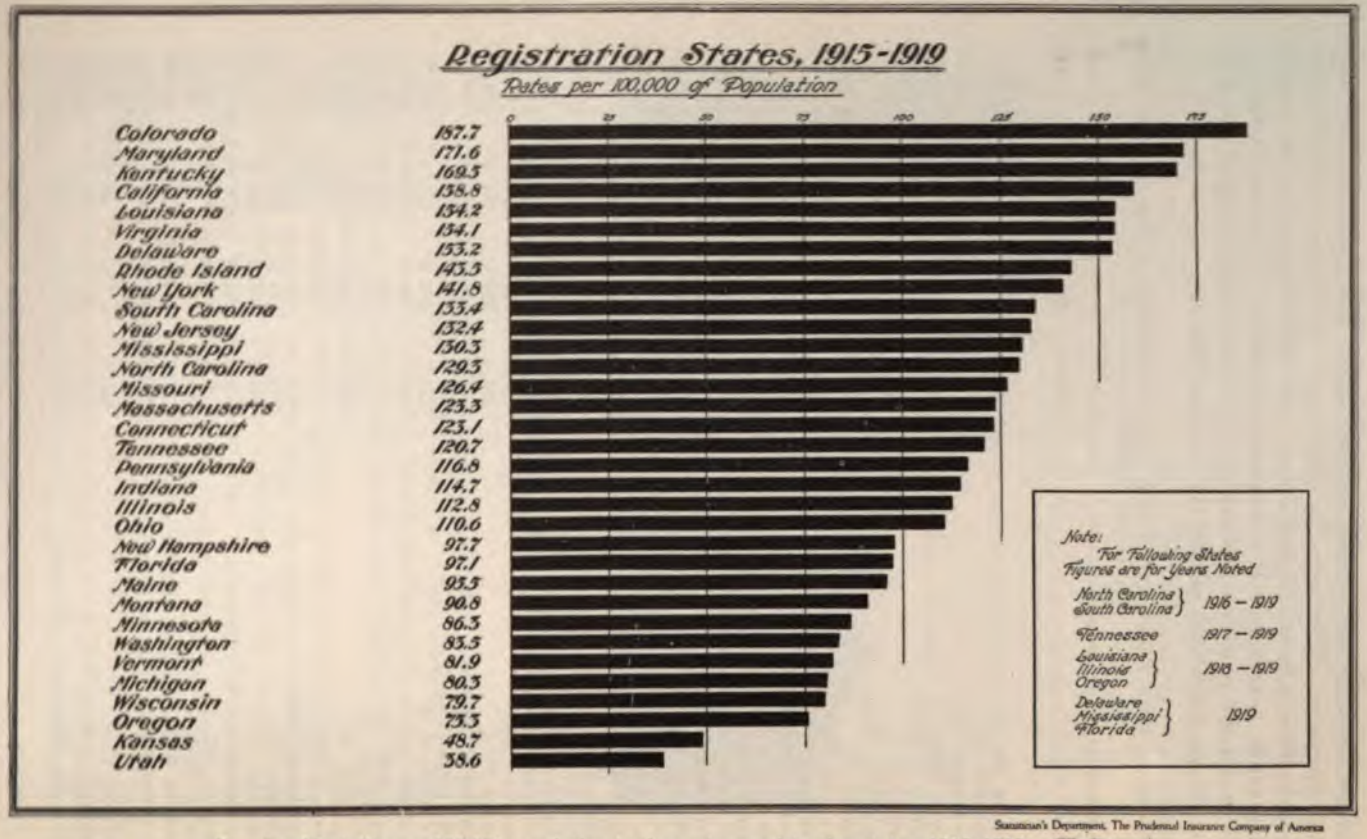


FIG. 8.—Mortality per 100,000 population in the various registration states, 1915–1919. (Hoffman.)

Social and Economic Factors.—There is no question but that infection with tubercle bacilli is to a large extent influenced by social and economic conditions; but it appears from available evidence that the development of phthisis is almost altogether dependent on these factors. Thus, we find among the so-called well-to-do, the cutaneous tuberculin reaction only rarely reveals hypersensitiveness in infants and children. Schlossmann even says that a positive skin reaction is hardly ever found in the children of his rich clientele, indicating that they are free from infection. The experience of American physicians appears to be the same effect, though we do not have data about inoculation of a large series of well-to-do children in this, or any other, country. It is, however, a rule among pediatrists to place great reliance on the tuberculin test in children. That this is justified in the case of infants of prosperous parentage may be true, but whether in older children a positive skin reaction is exceptional is open to question. When children attend school, and later when they go out into the world, meeting all sorts and conditions of men, they are no longer sheltered against infection, and most of them, in fact, do become infected sooner or later.

The high proportion of positive reactions obtained among children and adults in rural districts in Germany and Scandinavia, where infection has taken place despite the absence of known open cases of tuberculosis, and even where bovine infection could be excluded, appears to confirm this view. In fact, it is very rare to find an adult in a large city who does not show a positive skin reaction to tuberculin, irrespective of his social or economic condition.

Among the millions of proletariat in large modern industrial cities infection appears to be most rampant. Reliable tests—autopsies and tuberculin—have shown that very few escape infection, and the clinics, sanatoriums, and hospitals for tuberculous patients derive their clinical material mainly from these strata of population. A study of the mortality-rates also shows that these are the people who are most likely to succumb to tuberculosis. One has only to glance over the maps of New York City prepared under the auspices of Herman M. Biggs to be convinced that poverty and tuberculosis go hand-in-hand. The blocks inhabited by the rich show exceedingly few deaths from this disease, while those inhabited by the artisans, the laborers, and the poor—the "slums"—are appallingly studded with cases of phthisis. Poverty, filth, and overcrowding may act by favoring the spread of infection, or by reducing the inherent resisting powers.

Illustrations from other cities are not wanting. In Hamburg the death-rates from tuberculosis are in inverse ratio to the amount of income tax paid by the various groups of population. In Paris, Bertillon found that in the very rich district Élysée the mortality from tuberculosis is the least in the city; it is somewhat higher in the rich Opera district; higher in the very well-to-do district Luxembourg; higher yet in the well-to-do Temple district; very high in the poor Reuilly district, and highest in the Twentieth Arrondissement, where

the inhabitants are exceedingly poor. In Glasgow, according to Glaister, the mortality is higher among families living in one-room apartments than in those who live comfortably in several rooms. In Edinburgh, A. Maxwell Williamson[1] found that the number of cases of tuberculous disease increases in proportion as the house accommodations become limited. "Pulmonary tuberculosis is a disease which in 70 to 80 per cent of cases occurs in houses of three rooms and under; the number of cases is larger in two-room houses than in three; larger in houses of one room than in two; and the number of cases of the disease increases almost in direct proportion to the number of small houses in any district or ward of a city." The relation of phthisis to overcrowding is seen clearly in the industrial cities of the United States.

Similar investigations as to the relations of wages to morbidity and mortality of tuberculosis have shown that higher wages mean less of the disease (see p. 89). The experience of life insurance companies is to the effect that industrial policyholders, who pay small weekly premiums, are more likely to succumb to the disease than those who hold "ordinary" policies paying annual premiums. In Europe it has been observed that the larger the amount for which the person is insured, the less likely he is to succumb to tuberculosis.

The influence of poverty, with its concomitant poor nutrition, on the incidence of tuberculosis has been demonstrated recently in the countries affected by the war, directly or indirectly. The mortality increased wherever the cost of living went up—in Germany, Austria, France, England, etc., and also in the Scandinavian countries, in Holland, in Brazil, and Argentine. Scarcity of nourishing food, and its high cost, producing undernutrition even in those who ordinarily have plenty, is undoubtedly the agent. In Germany, and especially in Austria, the tuberculosis mortality rose to an appalling degree during the World War, and reached its apex during the eighteen months following the conclusion of peace. But as soon as economic conditions improved, food becoming less scarce, especially as soon as fat was procurable, a remarkable and prompt improvement took place and the death-rates declined, tending toward those that prevailed before the war.[2] In England and Wales the following figures, taken from the Registrar-General's Reports, show the effects of the hardships of the war on the tuberculosis mortality:

MORTALITY PER 1,000,000 FROM PULMONARY TUBERCULOSIS.

	1912–14.	1915.	1916.	1917.	1918.	1919.
Males	1179	1487	1624	1885	2077	1116
Females	849	912	913	969	1055	842
All	1005	1185	1252	1406	1543	971

[1] British Jour. Tuberc., 1915, **9**, 111.

[2] See especially Selter and Nehring, Ztschr. f. Tuberkulose, 1921, **34**, 1; Rosenfeld, Die Aenderungen der Tuberkulosehäufigkeit Oesterreichs durch den Krieg, Vienna, 1920.

The tuberculosis mortality increased between 1915–18, but sharply declined so that in 1919 it came down to a little below that which obtained in 1912–14, before the war. Similar conditions were observed in the city of Paris during the Siege in 1870–71 in an even more accentuated form.

The slums of large cities contain "lung" blocks which have been pictured in such sombre colors in the popular tuberculosis literature. Of course, the bad housing conditions are responsible to a large extent. But it must be remembered also that "a slum is not constituted solely of broken-down houses, but also of broken-down occupants, and it is perhaps easier to remedy the one than the other," says John Glaister.[1] Moreover, the tuberculous, unable to earn a living, are more likely to move into cheap, *i. e.*, unsanitary, dwellings. This is a factor which is not generally appreciated when slums and "lung blocks" are spoken of.

Thus, we have a vicious circle in the economics of tuberculosis. Poverty brings about congestion and overcrowding, enhancing the chances of massive infection; it also compels its victims to work in unsanitary factories, mills and workshops and at trades which are dangerous in this regard. The vitality is depressed and the powers of resistance reduced as a result of insufficient and improperly prepared food, so that infection more often terminates in phthisis than among those who are higher in the social scale.

However, that the well-to-do and rich do not escape is evident when we glance into the modern private sanatoriums, which derive their clientele from those who can pay more than fifty dollars per week, not including medical attendance. The resorts in Europe are also filled with rich consumptives, as can be seen in Switzerland and the Riviera. Of course, this shows that not all well-to-do individuals live wisely, even though they can well afford to do so.

Influence of Age.—In considering the influence of age on the incidence of tuberculosis we must again differentiate tuberculous *infection* from *morbidity* and from *mortality*, and also the various forms of the disease.

The newborn infant is free from tuberculosis, as we have shown; infection takes place during the lifetime of the individual who is exposed to the bacilli. We have already seen that those living in a tuberculous milieu do not escape, and during the first year about 15 per cent are infected; during the first five years, about 50 per cent, and at the age of fourteen, over 80 per cent are infected. Even children of non-tuberculous parentage are infected with tuberculosis to the same extent as those of tuberculous stock, but not at such an early age, and when reaching adolescence the difference is not so pronounced as would be expected *a priori*.

The morbidity from the disease is greatly influenced by age. During the first two years of life tuberculosis is very frequently encountered

[1] Practitioner, 1913, **90**, 344.

in the form of acute miliary tuberculosis, and tuberculosis of the joints, bones, and glands. Between two and ten years of age we mostly find the milder forms of osseous, glandular, and articular tuberculosis,

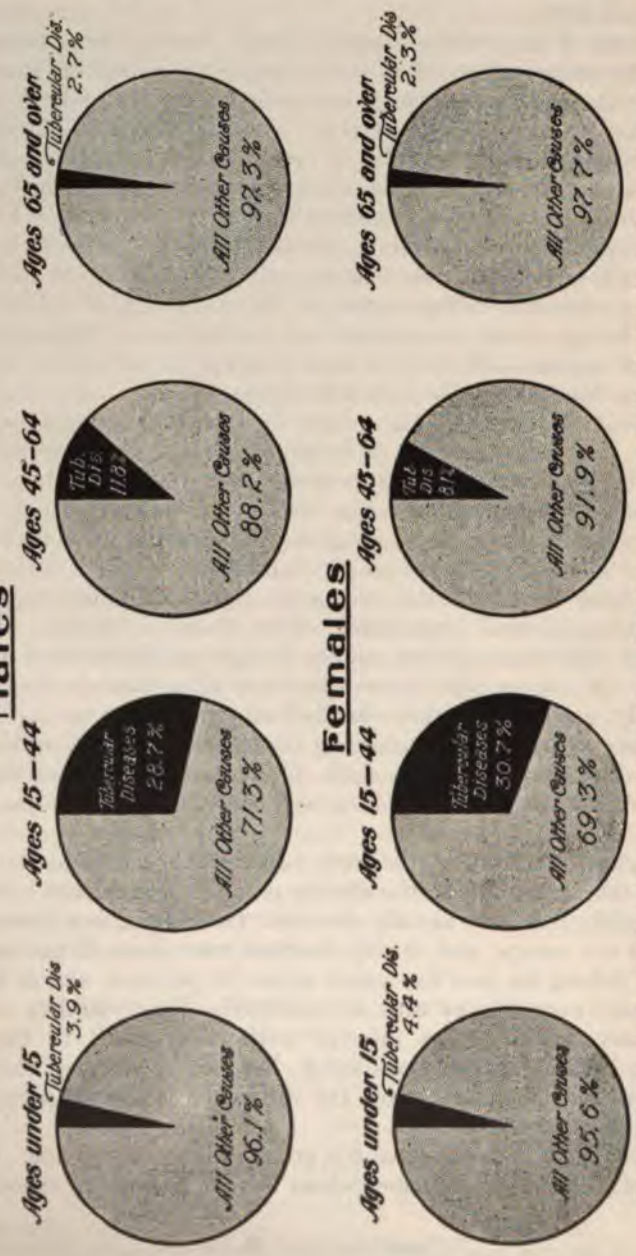


FIG. 9.—Proportional mortality from tuberculous diseases, by divisional periods of life, United States Registration Area, 1909–1913. (Hoffman.)

and chronic pulmonary tuberculosis is very rare. Only after the age of ten does the latter form of tuberculosis make its appearance, and after fifteen years of age it becomes the menace of society—the proverbial "white plague"—causing more misery than any other disease.

The disease is, however, for lack of reliable morbidity statistics, best gauged by a study of the mortality-rates. From the table below it is seen that there are two maximums of mortality. The first during the first two years of life; while beginning with the third year, tuberculosis becomes a very infrequent cause of death until the tenth year is reached, when it again begins to rise, reaching its full height at twenty years, and keeps at that high level with slight fluctuations until sixty years, when there is again a slight decline.

MORTALITY FROM TUBERCULOSIS IN THE REGISTRATION AREA OF THE UNITED STATES PER 10,000 LIVING AT THE GIVEN AGE AND SEX, 1910–1913.

Age.	Pulmonary tuberculosis.		All other forms of tuberculosis.	
	Males.	Females.	Males.	Females.
0 to 1	6.73	5.68	13.76	12.14
1	4.72	4.00	11.78	10.64
2	2.14	1.97	6.13	5.53
3	1.44	1.41	3.95	3.84
4	1.00	1.16	2.90	2.78
5	0.97	0.94	2.10	1.54
6	0.92	0.84	2.01	1.37
7	0.85	1.19	1.83	1.95
8	0.63	1.26	1.36	2.07
9	0.98	1.31	2.11	2.14
10 to 14	1.22	2.94	1.15	1.35
15 to 19	7.96	11.09	1.72	2.09
20 to 24	16.27	17.66	2.10	2.26
25 to 29	18.98	19.33	2.12	2.10
30 to 34	21.70	18.62	2.08	2.01
35 to 39	23.13	16.22	2.09	1.89
40 to 44	23.47	14.25	2.07	1.69
45 to 49	23.32	11.99	2.02	1.63
50 to 54	21.68	11.19	2.04	1.63
55 to 59	22.99	11.80	2.47	1.96
60 to 64	22.13	12.39	2.56	1.92
65 to 69	21.00	14.25	2.45	2.22
70 to 74	20.11	15.87	2.68	2.37
75 to 79	18.02	16.07	2.41	2.70
80 to 84	13.64	13.24	2.02	2.20
85 to 89	12.48	10.23	2.38	2.23
90 to 94	9.71	6.58	1.21	1.25
95 and over	10.37	6.71		1.52

It is thus clear that *the rate of infection with tuberculosis does not follow closely the rate at which the disease kills.* As shown in the table on page 71, infection begins during the first year of life, keeps on increasing during every subsequent year until at the age of twenty very few individuals are found who have escaped it. The mortality is comparatively high during the first year of life, but then declines, so that between three and twelve years, just the period when most infections occur, the number of deaths is the least, and only after the fifteenth year does

the mortality rise to its highest point, and keeps at it throughout life. The bearings of these facts on the problems of phthisiogenesis and prophylaxis will appear in other sections of this book.

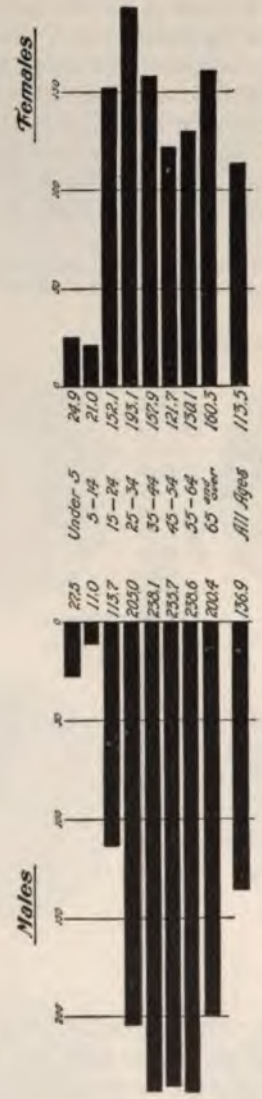


FIG. 10.—Mortality from pulmonary tuberculosis per 100,000 population in the United States Registration Area, 1915–1919, according to age and sex. (Hoffman.)

Influence of Sex.—From the table on page 85 we find that during the first six years of life the mortality from pulmonary tuberculosis is somewhat, though not very materially, less among females than among males. After the sixth year the rates among females are higher

than among males of the corresponding age groups. Between fifteen and thirty years of age the difference in favor of the males is striking. After thirty years the females again show lower mortality-rates which keep up until the end of natural human life. The total mortality is less among females than among males, a fact which has been observed in all countries where vital statistics are available. In New York City the mortality from phthisis in 1920 was: Among the total population 11.9 per 10,000; among males 14.9, and among females only 8.5.

Various explanations have been offered for this disparity in the mortality from phthisis between the two sexes. It has been suggested that the more hazardous occupations, in which men are mainly engaged, reduced their resistance, and predisposed them to phthisis; or when

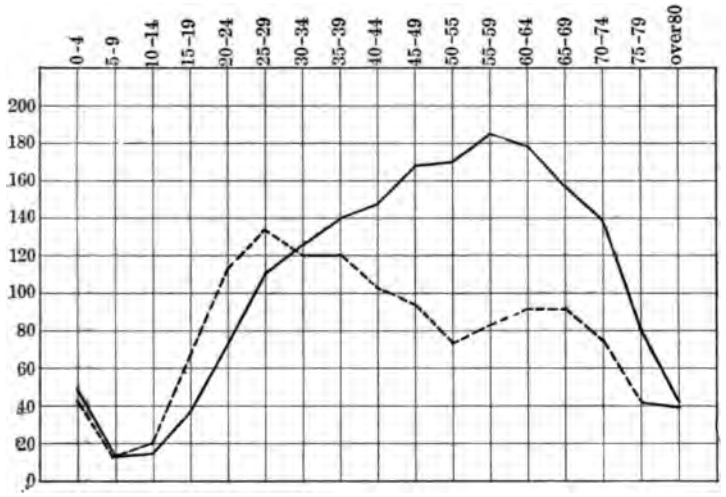


FIG. 11.—Death-rates per 100,000 population by age and sex in the Commonwealth of Australia for the years 1909–1913 (all forms of tuberculosis). Males, ————; females, ...

becoming sick with the disease, the chances of recovery are less in the case of men who have to work for their support, as well as for those depending on them. But during the ages of fifteen to forty-five, when menstruation, pregnancies and lactation undermine the resisting powers of women, it would be but natural that the mortality from phthisis should be high among them. Vital statistics in some countries seem to support this view, but in the United States and in the Commonwealth of Australia (Fig. 11) the higher mortality among the women keeps up only until the age of thirty, when it again declines as compared with the men.

It appears to me that the higher mortality from phthisis among women between fifteen and thirty in the United States is to be attributed to the large number engaged in gainful occupations. This is con-

firmed by the census returns showing that among all classes of population, male and female, ten years of age and over, without regard to occupation, the proportion of deaths from tuberculosis is 56 per cent

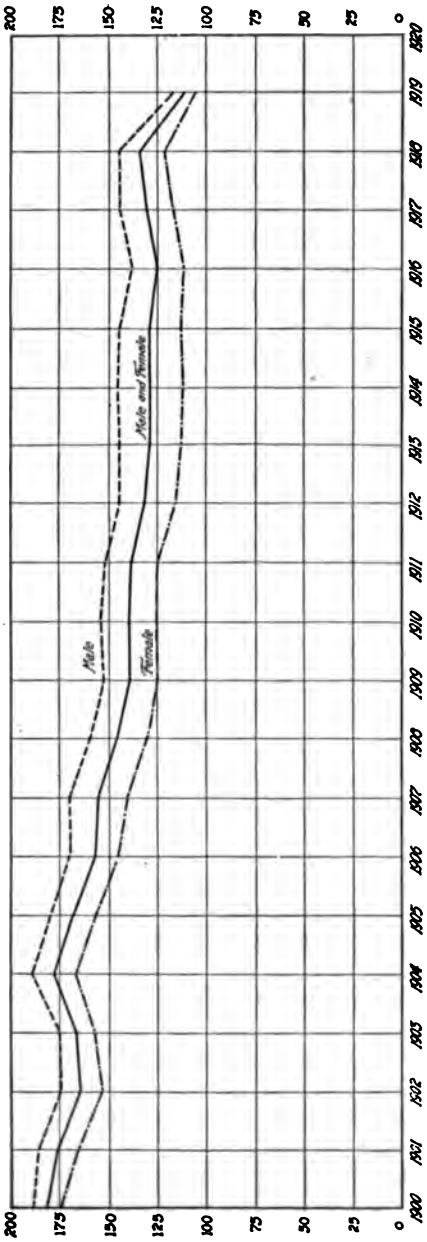


FIG. 12.—Decline in the mortality from pulmonary tuberculosis, 1900–1920, in the United States Registration Area. (Hoffman.)

males, and 44 per cent females. When women enter gainful occupations to earn a living, as B. S. Warren[1] has shown, the proportion is reversed and the difference much greater. Thus, among salesmen tuberculosis constitutes 15.8 per cent of all deaths, as against 31.1 per cent among saleswomen; among silk-mill weavers, men 19.7 per cent and women 38.3 per cent; among woollen-mill operatives, males 22.2 per cent and females 29.2 per cent; clerks and copyists, males 29.2 per cent and females 31.8 per cent; and boot and shoemakers, males 13.3 per cent and females 31.8 per cent. It thus appears that it is more a problem of industrial conditions than of sexual differences. In fact, women do not bear hard work under deleterious conditions as well as men, and succumb to phthisis in greater numbers when, in addition to exercising their physiological functions, they become bread-winners.

Since women entered industrial occupations, their mortality from tuberculosis has greatly increased. Thus, in Stockholm, the mortality from tuberculosis since 1881 has been in women only two-thirds that of men. E. Lindhagen[2] shows that between fifteen and twenty years of age the death-rate in women has, however, increased by 18 per cent, while that of men has been reduced by 12 per cent. During the World War there was noted an increase in the tuberculosis rates in females much more intense than that of the males. Conditions in England are given in the table on page 82.

In the Netherlands similar conditions have been observed. B. H. Sajet[3] shows that in cities the tuberculosis death-rates have increased since the war from 154.4 per 100,000 in 1913 to 175.9 in 1916. The mortality has, however, not increased materially among young men in the cities, but there has been noted a great increase among the women between twenty and thirty years of age, *i. e.*, among those of working age. This is, perhaps, best shown by conditions in Vienna, where the effects of the war have been most disastrous. According to Sigismund Peller[4] the mortality from tuberculosis before the war, in 1913–14, as compared with 1919, after the war was as follows, per 10,000 population:

	Men.	Women.
1913–14	35.5	24.8
1919	54.5	52.3

The increase in the mortality of the women is thus seen to have been 110 per cent, while of the men, 54 per cent. Similar conditions were also observed in other belligerent countries, and in those in which the labor market was affected by the war, though not to such an appalling extent as in Austria, Serbia, Poland, etc. It was not only the reduced food supply and the increased cost of living that was responsible for this increase, but also the fact that women, who

[1] Tr. Nat. Assn. Study and Prevent. Tuberc., 1913, **9**, 153.
[2] Hygeia, 1918, **80**, 497.
[3] Nederl. Tijschr. ven. Genaec., 1917, p. 1859.
[4] Wien. klin. Wchnschr., 1920, **33**, 906.

formerly were idle, or engaged in less dangerous trades, had to go to work at all kinds and conditions of labor, and thus their mortality from the most important of industrial diseases increased.

Mortality-rates from Pulmonary Tuberculosis.—It is impossible at present to give with certainty the extent of tuberculous morbidity in any population. Even in cities where registration of this disease is compulsory, the data collected in this manner are not complete, and we do not know the exact number of persons suffering from active tuberculosis. The statistics published by certain benevolent and industrial societies are also inconclusive because they concern only certain groups of people, and the results cannot be applied to the general population. Attempts have been made to ascertain the morbidity-rates from tuberculosis by multiplying the number of deaths occurring in a given region by the average duration of the disease. Thus, there annually occur about 150,000 deaths due to tuberculosis in the United States; in Germany, over 100,000; in France, 70,000; in England and Wales, over 50,000, etc. But attempts at multiplying these numbers by the number representing the average duration of the disease and thus finding the actual number of sick have met with failure because there is no agreement as to the average length of phthisis. Indeed, it has been estimated at from one to ten years by different authors.

The extent of the disease is therefore best gauged by the number of deaths it causes in a given population. The table on page 75 gives the mortality per 100,000 population in different countries. When in connection with these figures we bear in mind that one-third of all the deaths during the prime of life, between fifteen and forty, are due to tuberculosis, of which over 90 per cent is phthisis, we realize the enormity of the problem presented by tuberculosis and the reason why it has been considered the most important of diseases with which humanity has to cope.

Statisticians are, however, inclined to question the accuracy of the tuberculosis mortality statistics. Some state that many persons dying from other pulmonary diseases, notably bronchitis, pneumonia, typhoid, cerebrospinal meningitis, influenza, etc., as well as many other diseases, which occur in consumptives as often as in others, are reported as having died from these diseases, though the real cause of death was undoubtedly phthisis. This point is well illustrated in the mortality-rates in Italy. During 1896–1901 only 1060 per million died from tuberculosis in Italy as against 1911 in Switzerland. But in Italy during the same period there were reported as having died 2032 from bronchitis, 2031 from pneumonia, and a total of 4641 deaths per million from various diseases of the respiratory organs. In Switzerland during the same period the rates were: Bronchitis, 1092; pneumonia, 1525, and all respiratory diseases, 2828. Similar figures may be culled from the Registrar's Officers' reports in many other countries. This is also to be seen from the fact that in cities in which compulsory registration of tuberculous patients is enforced, a large proportion

who are reported tuberculous are in the end certified as having died from other diseases, which is undoubtedly true, because tuberculous patients are liable to other fatal diseases, but still, while alive, they were tuberculous and sources of infection. It has been my observation that in populations in which so-called "industrial insurance" is commonly taken out by the poorer strata of the people, tuberculosis is often not given as the cause of death, because it may interfere with the collection of the death claims from the insurance companies. It is also a fact that since tuberculosis has become an actual stigma, some deaths due to this disease are returned as having been caused by other diseases with a view of sparing the families the feeling of "tainted blood."

The differences in the mortality-rates for the various countries are due to diverse causes, mainly the intensity of concentration of population in cities, the character of the occupations pursued by the people, and other factors which have already been discussed.

Decline in the Mortality from Tuberculosis.—Another point brought out by the figures in this table is that the mortality from tuberculosis has been declining in nearly all countries where statistics are available, excepting in Norway, Ireland, Serbia, Spain, France, Italy, Japan, Hungary, etc. This decline is of great significance, and if the exact causes were ascertained we might be in a position to accelerate it, so that ultimately the disease could be stamped out altogether.

In England the decline in the tuberculosis mortality can be traced back for 150 years. "In the years 1743–53," says Arthur Ransome,[1] "when, as Ogle says, 'there were fairly accurate transcripts from the parish registers, the proportion of deaths was rather more than one-fifth; and, in the first returns of the Registrar-General, in 1838, in London, it was 1 to 6 or 8.' In other words, the rate per thousand deaths in the former period was about 200, and in the latter about 148. Hence, in the middle of the eighteenth century, phthisis must have been still more common than in 1838; and then the diminution in the mortality from the disease must have been proceeding steadily, at about the same rate as that observed in the earlier years." A glance at Fig. 14, showing the mortality in 1851–70, as compared with subsequent decennia to 1910, proves conclusively that the mortality has declined. The same is true of Scotland, Australia, Germany, Austria, etc. For the United States Frederick L. Hoffman's[2] statistics (see Fig. 13) tend to show that the mortality from tuberculosis in New York, Philadelphia, Boston, etc., has been constantly declining during the past one hundred years.

What are the causes of this decline in the tuberculosis mortality? All authorities agree that it is mainly due to the causes which have been operative in reducing the general mortality; in banishing, or abating, the malignancy of most other infectious diseases. Among

[1] Tr. Epidemiol. Soc., London, 1904–05, **24**, 259.
[2] Tr. Nat. Assn. Study of Prevent. Tuberc., 1913, **9**, 101.

these factors are largely to be considered the improvements in the sanitary and hygienic conditions under which the bulk of the people live at present. It is also to be considered that modern factory legislation, the improvements in the economic conditions of the people,

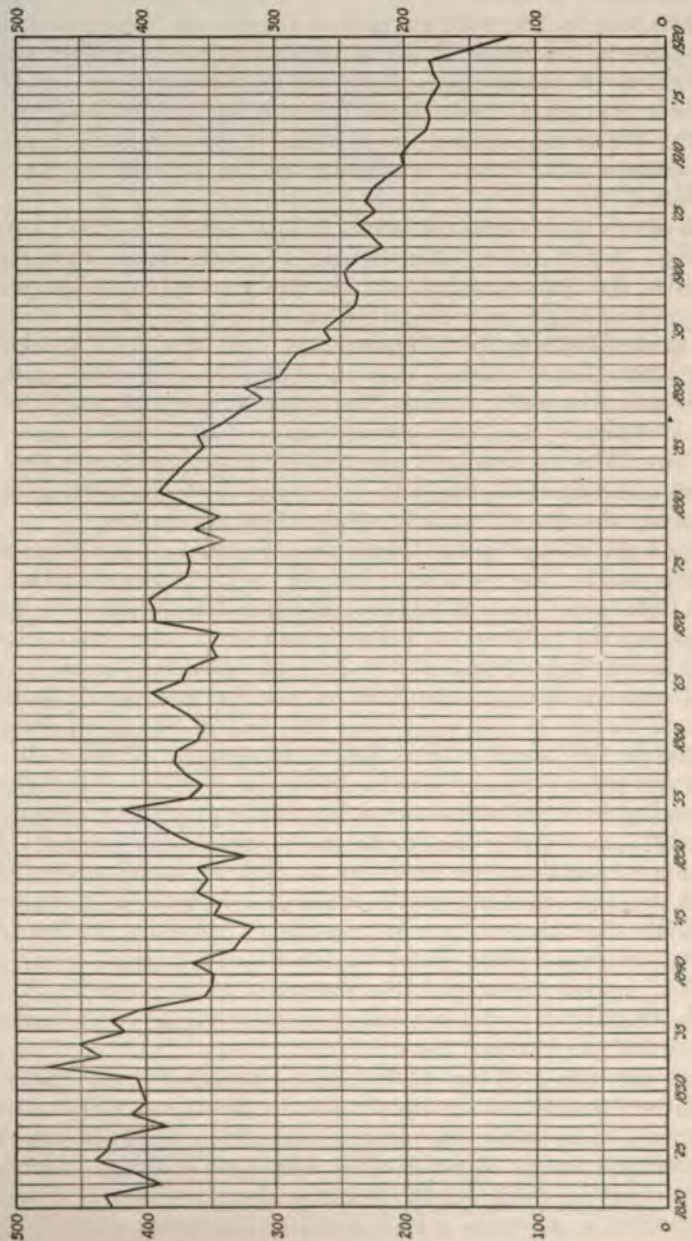


FIG. 13.—The decline in the mortality from tuberculosis in New York, Philadelphia and Boston, 1820–1920. (Hoffman.)

the shorter hours of work, etc., which are characteristic of the present, as compared with conditions during the first half of the nineteenth century, have been instrumental in reducing the general mortality and phthisis as well. Wages have been increasing, and the food consumed by the working people of today is much superior to that which they could afford fifty or one hundred years ago. The distribution of food, as well as its preservation, precludes famines at present. An increase in the tuberculosis-rates is often observed during and after famines.

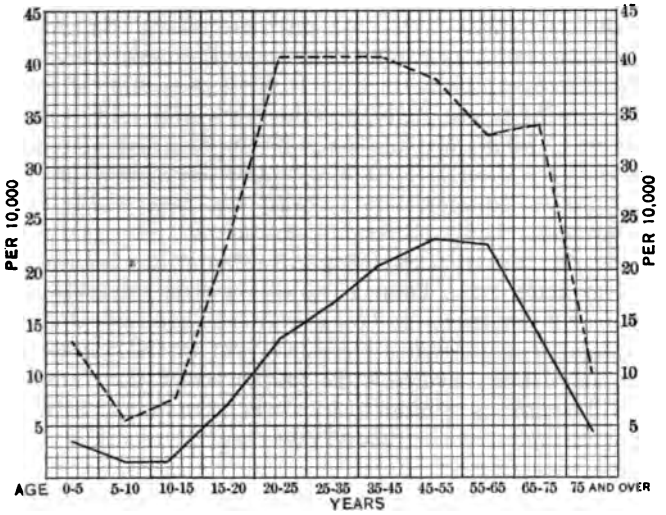


FIG. 14.—Mortality from phthisis by age groups in England and Wales per 10,000 living, showing the decrease from 1851 to 1912. Dotted line, mortality during 1851–1856; black line, mortality in 1912.

The Effect of the Special Campaign against the Spread of the Disease.—Most authors, when speaking of the reduction in the tuberculosis mortality, point at once at the special measures which have been taken to combat this disease as the sole factor in this direction. In fact, the figures compiled in the tables on pp. 76 and 79 are always brought forward in proof of the effectiveness of the antituberculosis campaign which has been so aggressively waged.

But careful studies of the available statistical data have not sustained this contention. In England, where the decline has been more pronounced than in any other country, it has been shown by competent statisticians that such is not the fact. Karl Pearson[1] points out that, examining available data, it appears that the death-rates from phthisis are steadily increasing as we go backward to 1838; according to Arthur Ransome even as far back as 1743, as was mentioned above. Now, this could not go on indefinitely because if it did, every individual five

[1] **The Fight against Tuberculosis** and the Death-rate from Phthisis, London, 1911, p. 9.

hundred years ago must have died in England from phthisis. There was assuredly a time in England when the phthisis rates were rising, just as they have recently been falling. "We have to stretch," says Pearson, "our ideas of time a little and we should realize the possibility of a typical epidemic curve in the frequency of phthisis. Indeed, the mortality from phthisis in England has been declining since 1838, *i. e.*, long before any special measures had been taken for the control of the disease, or segregation of the sources of infection—tuberculous human beings and animals—had been attempted."

Data from other countries, especially where the disease has become a menace during recent years, confirm these views. During the first half of the nineteenth century there were isolated areas in Europe where tuberculosis was rare, but with the segregation of the population in cities during recent years, and the introduction of modern industrial conditions, the disease has made its appearance, and rages there with greater vigor than in countries where the disease has appeared before. Thus, the tuberculosis mortality has been rising in Ireland, Norway, Sweden, Serbia, Bulgaria, Roumania, Hungary, Portugal, Italy, Japan, etc., during the very period that it has been declining in England, Germany, etc. There is no doubt that the measures taken for the control of the disease in Norway are as aggressive and advanced as those taken in neighboring Denmark, yet in the former the mortality-rates have been rising, while in the latter they have steadily declined. The same is true of France when compared with Belgium, and similar analogies can be made between other countries, or various regions of any single country.

The differences in the mortality from tuberculosis in the various cities in this country, as shown in the table on p. 79 point in the same direction. The various agencies engaged in the campaign against the spread of tuberculosis have not neglected the cities of Buffalo, Minneapolis, St. Paul, etc., yet the tuberculosis mortality has increased during the past fifteen years, while in others it has decreased.

It appears that *the mortality-rates from tuberculosis have been declining to the same extent as the general mortality from all causes, as has been shown clearly by many competent statisticians.* Professor Walter F. Wilcox[1] says that "to show that the campaign against tuberculosis is having its effects, it should be found that the death-rates from that disease are decreasing faster than the average for all other causes." But a test of this question with statistics for the mortality in the State of New York shows that the result is a negative one. "No influence of the special campaign can be traced in the figures. The condition in Michigan is similar to that in New York. In Indiana the number of deaths in each instance had decreased, but apparently the proportion of those from tuberculosis to all others has not." In New Jersey and Rhode Island, while the mortality from other causes has been

[1] Monthly Bulletin New York Board of Health, 1910, **26**, 85.

decreasing, that from tuberculosis has been increasing, so that the comparative proportion of the latter has risen. Pearson[1] has proved incontrovertibly that since the campaign has been waged in England against tuberculosis "the rate of fall in the death-rate from phthisis, instead of being accelerated, has been retarded." From careful statistical studies he arrives at the conclusion that "somewhere about 1915 the fall in the phthisis-rate which had been less rapid since 1895 would cease altogether and probably be followed by a rise. The next five years will show whether this is true or not."

Statisticians are not alone in this opinion. In a posthumous paper by Robert Koch[2] he states that the special measures taken for the control of tuberculosis, such as segregation of consumptives, the erection of sanatoriums, etc., are not to be taken as the sole factors which have been instrumental in reducing the mortality from tuberculosis during recent years. He says: "Many have correlated the decrease in the tuberculosis mortality with the discovery of the tubercle bacillus. It was stated that since proofs have been produced that tuberculosis is transmissible, greater care has been taken to prevent infection, while before the discovery of the tubercle bacillus, physicians, and with them the laity, denied the transmissibility of the disease. This assumption surely has something in its favor. At any rate, it is a striking fact that, with but few exceptions, the decline in the mortality began a few years after the discovery of this bacillus. But just these exceptions prove that the newly engendered fear of the dangers of infection is not the only factor operative in this direction, although we must give it a certain, and not an inconsiderable, amount of credit. Among German authors we often meet with the view that the recent social legislation, especially that concerning workmen's insurance, has been effective in reducing the tuberculosis mortality. To a certain degree there is some correlation in time between these two phenomena in Germany. But, inasmuch as in most other countries such laws have not been inaugurated and the decline in the tuberculosis mortality has taken place to the same extent as in Germany, this factor should also not be taken as a cause."

In this country we now hear similar opinions expressed. William Charles White[3] says: "We cannot possibly avoid the facts that in spite of all our labor our results are not what we might have expected on a right premise; for our reduction in morbidity and mortality from tuberculosis has not kept pace with the reduction in the general death-rate; and, further, our reduction in mortality was about as great before we started our present methods, and in proving how great the influence of our efforts has been we usually neglect all the influences that operated before we began, and new factors, such as the Mills-Reinecke phenomenon, and ascribe all good to our own work." Like-

[1] Biometrica, 1918, **12**, 347.
[2] Ztschr. f. Hyg., 1910, **67**, 1.
[3] Tr. Nat. Assn. Study and Prevent. of Tuberc., 1913, **9**, 80.

wise, Raymond Pearl,[1] one of the ablest biometricians in this country, says that the death-rates in Baltimore, calculated for the past ninety years, show the same tendencies to rise as was observed by Pearson to be the case in England. About the campaign against tuberculosis he has this to say: "When I see millions of dollars literally thrown away each year on charitable and public health activities—which every trained geneticist, if he used the analytical powers of his mind to the same good purpose that he does in his laboratory, could demonstrate to be futile, because of the limitations which known facts of heredity place on these well-meant endeavors—I am appalled and disheartened at the spectacle science permits to be made of itself."

Perhaps the best proof of the contention that the decline in the tuberculosis mortality during the past one hundred years has hardly, if at all, been accelerated by the campaign waged against this disease is to be found in the fact that *the number of persons infected with tubercle bacilli has not decreased.* We must bear in mind that modern methods of prophylaxis aim mainly at but one thing: The prevention of infection. In this we have utterly failed, as could be expected, considering the ubiquity of tubercle bacilli "carriers," and that hardly 5 per cent of "open" cases of tuberculosis have been isolated by hospitalization in even the most progressive communities. We have shown conclusively that over 90 per cent of humanity harbor tuberculous lesions in their bodies (see p. 70), indicating that our efforts at keeping these organisms from infecting humanity have hardly met with encouraging success.

Causes of the Decline in the Tuberculosis Mortality.—Despite our failure to prevent infection, there has taken place a very important decline in the morbidity, and particularly in the mortality, from tuberculosis during the past one hundred years in some countries, as England, the United States, etc.; and in others, like Germany, Austria, Holland, Switzerland, etc., during the past fifty years. As far as our present knowledge goes, we are not in a position to credit this decline to any single known cause; it is apparently due to many, and complex, factors which, with our present meager knowledge of phthisiogenesis, largely remain obscure.

Many, especially geneticists, suggest that tuberculosis is declining in frequency and fatality in countries in which it has been widespread for centuries by a process of natural selection. Pearson looks "upon the decline as a result of a pure selective process, the survival of people with an inherited immunity in various grades, and not with an acquired immunity." This view is not in conflict with our knowledge of the lack of inheritance of acquired characters. Persons with excessively susceptible constitutions die, mostly during the early years of life, leaving few or none who have inherited their weakly constitutions. In favor of this view is the hypothesis that the phthisical constitution

[1] Jour. Am. Med. Assn., 1920, **74**, 375.

is not a specific trait which predisposes to the disease, but a general predisposition, a point which is discussed in detail in Chapter IV. The facts presented above (see p. 73) about the deadliness of tuberculosis in communities in which it was not known before, also tend to confirm this view. Available epidemiological data tend to show that wherever people who have hitherto been free from tuberculosis come into tubercle laden surroundings, they succumb to the more acute and fatal forms of the disease, while most of those who have for generations been tuberculized are either not harmed by the specific germs, phthisis not developing after the vast majority of infections, or when this does take place, the disease manifests a strong tendency to pursue an exceedingly chronic course, or heals spontaneously in the vast majority of cases. This point has been discussed in detail in a paper by the writer[1] on "Tuberculization and Immunization;" more recently Bushnell[2] has published an excellent work on this subject. It seems that after contact with the tubercle bacilli for several centuries, the human being adapts itself to the conditions created by these micro-organisms, which can live in symbiotic relationship with the host.

Careful studies of the economic and social conditions in the various European and American countries in which statistical data are available, tends to show that there is a pronounced correlation between urbanization, *i. e.*, concentration of large masses of population in cities, and the death-rates from phthisis. Wherever the process of urbanization is new, wherever modern industrial conditions have only recently been introduced, and large numbers of rural people have been attracted to cities, the death-rates from phthisis have been rising. This is the case in Japan, Norway, Sweden, Ireland, Serbia, Roumania, Bulgaria, Russia, Austria, Italy, France, etc., in which countries the death-rates from phthisis have either increased during recent years, or have failed to decline perceptibly. On the other hand, in England, where industrial development was already on a high scale at the beginning of the nineteenth century, it was at that time that the high phthisis mortality occurred, and it began to decline with the increased adaptation of the population to city life. In line with this urbanization can be considered the conditions of the negroes in the United States. The phthisis mortality among them is higher than among the whites. Though it cannot be said that very efficient measures are taken by them to prevent dissemination of the disease, there has been found a strong tendency toward a reduction in the death rates. Thus, in Baltimore, John W. Fulton found to his amazement that "both races gained against tuberculosis, the whites at the rate of 30.8 per cent, and the negroes at the rate of 24.5 per cent, in the decade of 1904–1913."

While it cannot be said that social and economic conditions are altogether responsible for the incidence, or lack, of tuberculous disease, there seems to be no doubt that within certain limits they are the determin-

[1] Fishberg: New York Med. Jour., 1914, **100**, 497, 566.
[2] A Study of the Epidemiology of Tuberculosis, New York, 1920.

ing factor in a large proportion of instances. There can be made out a good case in favor of high wages, which have as their concomitants superior housing conditions, better nourishment, etc. (see pp. 81 and 89). Indeed, in countries in which social and economic conditions of the workmen are superior, the mortality from tuberculosis is low and keeps on declining. With the improvement in the economic conditions of the British and American laborers during the past century, the tuberculosis mortality has decreased. With the misery brought about by the World War, the tuberculosis mortality in Austria, Germany, Poland, France, England, etc., increased to an appalling degree. The increase in the female mortality from phthisis observed in some countries, warring and neutral (see p. 89), during that war, may be attributed to some degree to the entry of women into factories in large numbers. In line with this is the fact that while the mortality from this disease increased during the World War in most of the participating countries, in the United States, where food has been abundant, it declined. However, an association between the economic conditions and the tuberculosis death-rates is suggested in the fact that immediately preceding the rise in mortality during 1914–1915, there was a period of serious unemployment, and that in 1917–1920 wages kept pace with living costs and the demand for labor was extraordinarily great.[1] The sharp decline during 1920–1921 has been attributed to another factor: In 1918–1919 the epidemic waves of influenza carried off many persons who would have died within the two succeeding years. In the Report of the Registrar-General of England and Wales, Stevenson is inclined to explain the drop of the tuberculosis mortality in that country to the same cause.

At the present state of our knowledge, which is quite incomplete, the decrease recently observed in the mortality rates from tuberculosis can be attributed, in great part, to two factors: Mutual adaptation of the parasite and the host, and improved social and economic conditions of the great masses of population. The first has been brought about mainly by a process of natural selection, the more susceptible succumb to the tubercle bacillus. The second apparently depends on the improved social and economic conditions of the great masses of population in modern Western European and American urban centers. It appears that in well-fed, and properly housed individuals tuberculous infection, which is inevitable in nearly all who live in modern cities, is less likely to develop into the disease, phthisis, than in those who are undernourished and famished.

Brownlee's Three Types of Phthisis.—In a very careful statistical study of the incidence and mortality of tuberculosis in Great Britain and Ireland, John Brownlee[2] has arrived at the conclusion that *phthisis pulmonalis* is not a single disease, as is commonly understood, but

[1] See Public Health Reports, 1921, **36**, 1178.

[2] An Investigation into the Epidemiology of Phthisis in Great Britain and Ireland, Medical Research Committee, Special Report Series, London, 1918–1920, Nos. 18 and 46.

rather a group of diseases, in the sense as has been found the case with typhoid fever and bacillary dysentery. He brings strong evidence in support of his contention that there are three types of phthisis. the "young adult," "middle age," and "old age" phthisis.

The Young Adult Type.—The "young adult" type is the most important. It occurs mostly in young adults, twenty to twenty-five years of age. It is very much influenced by the geographical environment, being more common in districts exposed to the southwest wind, and above glacial clay than above glacial sand, and is less common in those districts in which there is a larger number of deaths from tuberculosis in children, where there is a high mortality from phthisis in "middle age," and in places where there is a considerable amount of tuberculous milk. Persons exposed to wind and wet, as seamen, dock laborers, agriculturists, etc., are more liable to succumb to this type of phthisis. It appears from Brownlee's statistics that the incidence of this type has no relation to the hygienic surroundings, or with overcrowding of population; it is associated rather with rural than with urban conditions.

The Middle Age Type.—The second type of phthisis has its incidence in middle age, killing most commonly between forty-five and fifty years. It is independent of the geographical and meteorological environment, of wind, and the nature of the subsoil, and is more common in those districts in which there is a large number of deaths from tuberculosis in children, and a large amount of tuberculous milk. It is especially common in persons pursuing certain occupations, mainly those associated with dust and silica. This type of phthisis is greatly influenced by hygienic conditions and density of population. Brownlee interprets these facts as follows:

"(*a*) Many persons infected with tuberculosis in childhood seem, after obtaining some degree of immunity, to go through life comparatively well, the organism of tubercle, however, remaining living though in a quiescent stage. As the strain of life increases, and the resistance falls, this chronic process seems, in a considerable number of cases, to flare up into an acute process, which results in subsequent death. This, however, would probably not account for more than two-fifths of the cases.

"(*b*) It is to be noted that the age at which middle-age persons die of tubercle is the same at which insanitary conditions have been shown to produce the most depressing effects, namely, between the ages of forty and fifty. Infection in middle-age would also seem to be common, especially among workers in silica."

The Old Age Type.—This type occurs mostly among persons fifty-five and sixty-five years of age, is especially common among miners of coal, lead, iron, etc., and among workers in slate quarries. It appears to be independent of hygienic conditions or the density of population, and no external factor has been found associated with it.

Brownlee attempts to offer two possible explanations for these

epidemiological peculiarities of phthisis. The first is that there are two distinct strains of the bacillus of tuberculosis. These different strains have each a special age distribution. One of the strains attacks in early manhood and possibly also in old age, the other in childhood and middle life. The one is associated with the country, and the other with the town. The second explanation offered is that the physiological response to attack by the organism may be different, according to the environment in which the individuals attacked have lived. From the first hypothesis it would appear that each type of organism confers a certain degree of protection against the other. An analogy is to be seen in the two distinct types of smallpox observed in England and attributed to distinct types of organisms; the same is apparently true of typhoid and paratyphoid. The second hypothesis is in agreement with the almost universal observation that mild infection with tuberculosis establishes some degree of immunity to the disease in those who survive. The result is that when the young adult stage of life is reached, the disease is absent. If, on the other hand, no immunity has been established when infection took place, the disease is often fulminant. Later, when the strain of life in cities begins to tell, namely, between forty and fifty years, those persons at the ages where the strain is most felt, tend to succumb. In the country the health of persons at these ages is good, and consequently death from phthisis at this age is rare.

The conclusions of Brownlee have great epidemiological bearings, and prophylactic measures, to be proved effective, must evidently be judged by their effect on the different types of the disease. The subject deserves further study in other countries.

CHAPTER IV.

PHTHISIOGENESIS. I.

PREDISPOSITION, ENDOGENOUS AND EXOGENOUS.

THE data presented in the preceding chapters have shown convincingly that tuberculosis is a transmissible disease, caused by the entry of the tubercle bacilli into the body. But it was also made clear that while no tuberculosis can occur without tubercle bacilli, infection with the germs is not invariably followed by that train of symptoms which we know as tuberculous disease. Indeed, *forty years of intensive study of the bacteriology of tuberculosis has demonstrated that only in a relatively small proportion of cases is infection followed by tuberculous disease; the majority of human beings bear this infection without any harm.* On this point nearly all authorities agree. As was already mentioned, the only difference of opinion among pathologists at present is whether as many as 95 per cent of civilized humanity show evidences that tubercle bacilli have been implanted in the tissues of their bodies, or merely 70 per cent. Just as the presence of meningococci in the rhinopharynx does not invariably produce meningitis, pneumococci, pneumonia, Pfeiffer's bacillus, influenza, etc., so do not tubercle bacilli produce tuberculous disease in all cases in which they have gained an entrance into some vital tissues, even though they may have succeeded in producing gross or microscopic changes in the structure of the organs.

Constitutional and Environmental Causes.—Succeeding in elucidating tuberculous infection, but failing to explain satisfactorily tuberculous disease, bacteriology is at present considered by many authors as incapable of clearing up completely the etiological problems of phthisis. This is not only true of tuberculosis but of nearly all other infectious diseases. "Today we know that to have identified the microbic agent of any pathological process," says Theobald Smith,[1] "is but the beginning of the solution of the immediate problem and that it answers but one of a long series of questions." During recent years thoughtful minds in the medical world have therefore again directed their attention to other factors, endogenous and exogenous, which are instrumental in the production of pathological processes. The problems of heredity and environment, susceptibility, predisposition, and immunity are studied a'ong new lines of research. Attempts are being made to unravel the mystery why when several persons are

[1] Science, 1921, N. S., **54**, 99.

exposed to infection with tubercle bacilli, and infected, some may become sick, while most remain in comparative, or complete, health; why in familial tuberculosis, a few descendants of phthisical parents will develop tuberculous disease and perhaps die as a result of it, while several others equally exposed to infection remain healthy; why of those who become sick, some, though very few, suffer from a very acute and rapidly fatal disease, like miliary tuberculosis, or pneumonic phthisis, while many others have a chronic, but progressive disease, like pulmonary tuberculosis of the common type; why in still others, the virus produces evident changes in the lungs and pleura, but the process is abortive, the patient and his physician knowing little, or nothing of the infection. Reasons are sought for the preference of the virus to attack in some people the lungs, in others the glands, joints, bones, or serous membranes of the chest, abdomen, or the cerebrospinal axis. We are seeking light on the problem why clinical manifestations of tuberculous infection are observed only in a small proportion of individuals who harbor tubercle bacilli, while the vast majority in whom we know that the virus has undoubtedly been implanted in the tissues, produces anatomical changes in structure, unmistakably recognized at the necropsy, yet these lesions healed spontaneously without producing any obvious symptoms.

Various explanations have been given for these obvious differences in the pathogenesis of tuberculosis. Some have maintained that the variability of morbid phenomena is due to differences in the type and the virulence of the tubercle bacilli. But we have already shown that nearly all the tuberculous disease in adults is due to the human type of bacillus, and so is extrathoracic tubercle in children. It is also agreed that a study of the virulence of various strains of the virus isolated from the many clinical forms of the disease sheds no light on the problem. Guinea-pigs, monkeys, and primitive human beings, when exposed to infection, and infected, almost always suffer from the acute forms of the disease. Civilized human beings, infected spontaneously, mostly suffer from the chronic forms of phthisis, and in the vast majority of cases tuberculous infection is but an adaptation of the parasite to the host, both of which live in symbiotic relationship with each other.

Attention has recently been turned to accessory non-parasitic causes of phthisis, among which there are many, including endogenous, such as heredity, anatomical and biochemical peculiarities of the individual, etc., and also exogenous, including environmental peculiarities, such as economic conditions including occupation, housing, nourishment, etc., and also the geographical milieu. As will be seen from the succeeding pages, the results have so far been very meagre, but when a bacteriologist of the magnitude of Theobald Smith is constrained to say that non-parasitic factors are necessary conditions in the origin of infectious diseases, and far outweigh the living agent in etiological significance, it is clear that they are worthy of intensive study.

HEREDITY.

Statistical Study.—For centuries it has been noted that in certain families tuberculosis reappears in successive generations; many tuberculous patients can trace the disease back to their ancestors and blood relatives. Statistics collected along these lines are plentifully available in medical literature, but on close analysis it appears that they are of little value in proving or disproving the hereditary transmission of the disease, or a predisposition to it.

The element of contact with "carriers," or sources of infection, is always disturbing. Moreover, the ubiquity of the disease, one out of every seven or eight deaths is due to tuberculosis, so that it may be found in every large family or its collaterals, would lead us to expect that most tuberculous patients may find some relative who has had the disease. Then it must be mentioned that facts pertaining to heredity are derived from statements of patients in answer to leading questions, and these are open to criticism. Even questions about their personal history are not accurately answered, as a rule. Our patients at the Montefiore Hospital nearly always state that they had measles during childhood, probably on the principle that everyone must have it. But very few say that they have had diphtheria, typhoid fever, typhus, scarlet, etc., although most of them come from Eastern Europe where these diseases are rampant, and hardly any attempt worth mentioning is made to check them by proper quarantine regulations, and very few indeed escape. Very few know the real cause of death of their parents; in fact, it would seem as if their parents were all immune to tuberculosis, considering that the patients do not mention it after questions are addressed to them on the subject.

In private practice, where we deal with a more intelligent class, we often find that the father is stated to have coughed, the mother had hemoptysis, etc., after a categorical answer that there has been no consumption in the family. On the other hand, we know how much suggestion, through leading questions suitable for a certain purpose, may bring out appropriate answers. Many patients are convinced that their blood is not by any means "tainted," that they "come from healthy stock," that "there has never been any consumption in the family," etc. Moreover, the few facts obtained from patients, intelligent and otherwise, refer to the incidence of pulmonary tuberculosis, while extrathoracic tuberculous lesions in parents, brothers, and sisters, are almost always forgotten; at any rate not mentioned. As a rule, only fatal cases are mentioned, while those who have had the disease but recovered, are forgotten.

Statistical proof of the hereditary transmission of tuberculosis, or a predisposition to it, will only be satisfactory when careful records of many patients are kept for several generations in which children of tuberculous parentage have succumbed to the disease despite the fact that they have been removed immediately after birth, thus

preventing exposure to infection through intimate contact. To be of scientific value, they should be based on detailed postmortem examinations of the parents and grandparents, and even of collaterals. Such data we do not have. The frequently cited statistics obtained in orphan asylums showing that thousands of children of tuberculous parentage failed to develop tuberculosis, are absolutely of no value in disproving either heredity, or infection of this disease. In these institutions children under fourteen years are usually kept, and at that age active pulmonary phthisis is exceedingly rare, as has already been shown.

For these reasons very little confidence can be placed in the statistical compilations of various authors to the effect that among their patients 25, 44.7, or 59.2 per cent have given a history of tuberculosis in the parents, grandparents, brothers, sisters, or collaterals. It depends a great deal on the zeal of the questioner to obtain points for the substantiation of his pet theory. Even the excellent statistical studies of Karl Pearson, Weinberg, Schlütter, and many others, are not at all convincing. In fact, Burckhardt[1] found that in non-tuberculous persons, tuberculosis in the ascendency is just as strongly represented as in the tuberculous, and that the disease in the father is just as frequent in both groups, while the frequent occurrence in the mothers, fathers, brothers, sisters, uncles, and aunts, can be easily explained by infection.

Raymond Pearl[2] has recently attempted to meet the various objections to the statistical data thus far published by an ingenious method. He argues that if heredity is a factor of importance in the etiology of clinically active tuberculosis it would be reasonable to expect that a tuberculous individual would have a larger proportion of tuberculous among his or her blood relatives, both direct and collateral, and in ascending and descending generations from the individual, than would a person who was not tuberculous. His study is based on data obtained from 57 family histories, involving over 5000 blood relatives. Of these 38 were tuberculous, and 19 nontuberculous. Though the number of families is small, they may be regarded as random samples of the working population of Baltimore. His results showed that taking all generations together, it appears that a tuberculous person has 7 per cent of his or her blood relatives tuberculous, whereas a nontuberculous person, chosen at random, has only 1.2 per cent of his or her relatives tuberculous, and the absolute number involved in the two samples being absolutely the same. In other words, tuberculous persons have nearly six times as many blood relatives tuberculous as have nontuberculous. But closer analysis of his data led Pearl to the conclusion that familial contact with active open cases is beyond question a factor in determining the incidence rate of clinically active tuberculosis.

It is thus clear that the statistical method thus far has neither

[1] Ztschr. f. Tuberkul., 1904, **5**, 29.
[2] Am. Rev. Tubercul., 1920, **4**, 688.

proved nor disproved the hereditary transmission of the disease. Moreover, "if we allow that 50 per cent of the offspring of those suffering from pulmonary tuberculosis eventually suffer from this form of the disease, owing to their inherited want of resistance we should, I think, expect a progressive increase in the total mortality provided we bear in mind the increase of population. But the reverse appears to be true" (Latham). On the other hand, the most learned apostle of the theory of heredity of tuberculosis, Karl Pearson,[1] has this to say: "We do not suppose tuberculosis to be handed down from parent to offspring any more than we should suggest that the wearing of spectacles is hereditary. We merely suppose that certain constitutions are more and certain other constitutions are less resistant to, say, pulmonary tuberculosis. It is immaterial whether the tuberculous diathesis is looked upon as an inheritance of susceptibility or an inheritance of resistance, for both are but grades in the scale of immunity peculiar to the individual. In a community wherein tuberculosis has been prevalent for many centuries we should anticipate that natural selection would steadily intensify their immunity by eliminating those with less resistance; the higher grades of resistance survive and are transmitted by heredity. In a community wherein the tubercle bacillus has not been introduced there will have been no selection to raise the average degree of immunity; there will, however, be many grades of susceptibility, and those will be inherited, whether or not they have been put to the test of an infected environment." Our study of the epidemiology of tuberculosis (Chapter III) has brought forth evidence in favor of this view.

BIOLOGICAL ASPECTS OF TUBERCULOUS HEREDITY.

The reappearance of tuberculous disease in several successive generations is by no means conclusive proof that the disease, or that a predisposition to it, has been transmitted by heredity. We find in coal miners anthracotic changes in the lungs through several generations; so long as they are engaged at that occupation. But no geneticist will agree that we deal here with biological heredity. Likewise, the social, economic, hygienic, and sanitary conditions and surroundings which were responsible for phthisical disease in the parents may be, and usually are, operative in the children who remain in the same social milieu. In these cases sociologists speak of *social heredity;* it is not a biological phenomenon. *Biological heredity implies the transmission of characters, or their physical foundations, which were contained in the germ plasm, or the parental sex cells, at the moment of fertilization.* Anything that may have affected the fertilized ovum, or the embryo, cannot be considered inherited, because intra-uterine infection, and germinative transmission of a disease, have nothing

[1] Lancet, 1920, **2**, 891.

to do with the problems of heredity, just as extra-uterine influences cannot be considered transmissible in the biological sense.

As physicians, engaged in the study and prevention of the disease we must consider possibilities of embryonic infection, irrespective of its relation to the problems of heredity in the strict biological sense.

1. The unfertilized ovum may be infected from the mother.
2. The semen or the spermatozoa, infected from the father, may bring along the virus when reaching the ovum.
3. Tubercle bacilli may infect the fertilized ovum.
4. The embryo may be infected by bacilli in the mother's blood.

Experimental investigations by Friedmann show that intra-uterine infection with tubercle bacilli is not impossible. This, to some degree, confirms Baumgarten's theory to the effect that tubercle bacilli may enter the blood stream of the fetus, remain dormant for a long period of years, to flare up again by intense multiplication when, for some reason, the natural resistance of the body fails. This form of transmission of phthisis cannot be considered germinative heredity in the strict sense of the word—it is actually infection of the fetus from the mother—yet it is important for the clinician, especially to one giving thought to prophylaxis, and the modern teachings of eugenics.

Baumgarten[1] based his theory mainly on experiments with tuberculous birds. It is well known that the progeny of tuberculous chickens is tuberculous even under conditions in which infection after the egg has been laid can be positively excluded. It has been demonstrated experimentally that the albumen of a fertilized egg may be inoculated with tubercle bacilli, and the evolution of the chick goes on as may be expected; but it develops tuberculosis after it is hatched. This has been observed by Baumgarten, Milchner, Gärtner, Maffucci, Koch, and others. *Intraovular infection* has also been observed in cattle, and it shows that infection of the ovum, after fertilization, does not invariably destroy it. It may keep on growing and evolve into a living organism, even though it develops tuberculous disease soon after birth. In human beings, there have been found instances in which localized, calcareous changes of some focus in the lungs were seen in newborn infants, showing that they had tuberculous disease in utero and that the lesions had healed.

Spermatogenic Infection.—We are in the dark as to how the bacilli reached the embryo in such cases. Some authors maintain that the semen may be infected, and the bacilli are carried by the spermatozoa. Spano, Porter, Friedmann, and others have, in fact, found tubercle bacilli in the semen of persons who died from acute miliary tuberculosis, and such as have suffered from tuberculous disease of the generative organs. It is important to bear in mind that persons with tuberculous disease of the testicles, seminal vesicles, prostate, etc., at times cohabit with the opposite sex, and pregnancy occurs quite

[1] Arb. a. d. Gebiet d. pathol. Anatomie u. Bakteriol., 1891–1892, vol. 1; Lehrbuch d. pathogenen Mikroörganismen, Leipzig, 1911, p. 710.

often in such cases. Indeed, Albrecht, Cavagnis, Maffucci, and others, have succeeded in infecting rabbits and guinea-pigs, with semen taken from bulls suffering from tuberculosis. Friedmann[1] injected an emulsion of tubercle bacilli into the vagina of rabbits immediately after they had been impregnated by the males. Subsequent observation showed that while the mothers remained free from disease, tubercle bacilli were found in sparing numbers in the seven-day-old fetuses, which were apparently not at all hampered in their evolution. In the newborn rabbits, whose mothers were thus treated, tubercle bacilli were found in various organs. These findings were taken as evidence to the effect that *spermatogenic infection*, *i. e.*, infection with tubercle bacilli brought along with the semen from a tuberculous father, is possible.

It appears, however, that things are not so simple. Semen contains tubercle bacilli only when the generative organs, especially the testicles and seminal vesicles, are affected by tuberculous disease, though Jani, Spano, Jackh, Nakarai, and Simmonds have found numerous tubercle bacilli in the apparently normal seminal vesicles of patients suffering from tuberculosis of the lungs. Rational consideration, however, leads one to the suspicion that all this is not sufficient proof that spermatozoa, or the ovum, are infected with tubercle bacilli. The size of the mammalian ovum and spermatozoön renders it extremely improbable that they should become infected with these germs. Indeed, it may be stated that no one has ever seen a spermatozoön, or an ovum, in which a tubercle bacillus could be discerned under the microscope. Moreover, if they were infected, they surely could not go on developing; even if they were not killed, they would undoubtedly be rendered sterile. The fact that semen is occasionally found to contain tubercle bacilli, as is shown by its potentiality to infect animals, does not prove that germinative infection ever takes place. Clinically we see very frequently that children begotten by tuberculous fathers, even those who have tuberculous lesions of the generative organs, are well developed; on the average, as well as those of apparently healthy parentage. Moreover, we know of no case reported in which a father with tuberculosis of the testicles, seminal vesicles, or prostate, had begotten a congenitally tuberculous infant, though Hildebrand reports a series of cases in which intercourse with men having tuberculous testicles was responsible for tuberculous disease of the female generative organs. Even conceding that the tubercle bacilli may implant themselves on spermatozoa, and then, at the time of fertilization, infect the ovum, it must be an exceedingly rare occurrence. We must recall that with each emission over twenty millions of spermatozoa are expelled, and that the one on which a tubercle bacilli has implanted itself should be just the one that fertilizes the ovum, is a rather remote chance. This mode of infection

[1] Ztschr. f. klin. Med., 1901, **58**, 2.

of the ovum may thus be left out of consideration when speaking of tuberculous heredity.

Placental Transmission.—Many diseases are often transmitted from the mother during pregnancy; smallpox, syphilis, leprosy, etc., are good examples of intra-uterine infection of the healthy fetus from a sick mother. That the placenta may harbor tubercle bacilli is well known; the frequent bacteriemia in phthisis explains it. Lehmann, Runge, Nowack, Auche, Chamberland, Warthin, Weller, and many others, have found tubercle bacilli in the human placenta. On carefully examining the histology of the placenta of phthisical pregnant women Schmorl and Geipe[1] found tubercle bacilli in 9 out of 20 cases. One of the 9 women had merely an incipient apical lesion. Schmorl estimates that 50 per cent of pregnant phthisical women have tubercle bacilli in their placentas. He maintains that tubercle bacilli may enter the placenta during any period of pregnancy, and in any stage of the disease, but that they are mostly found in the advanced stages of phthisis, and in acute miliary tuberculosis. The fetus may be infected from the mother during the act of birth, when vigorous contractions of the uterus may lacerate some of the less resisting parts of the placenta. But there is a possibility of earlier infection. It is, to be sure, only a remote possibility that the virus should directly enter the fetus, if it is at all possible, but it may be brought along with blood through the umbilical vein; or by way of the intestine after it reached the amniotic fluid and is then swallowed, or aspirated by the fetus. According to Andrews, Heller, Sitzenfrey, Rietschel, Ghon, and others, the amniotic fluid at times contains tubercle bacilli. In some cases they may infect the chorion and, by extension, tuberculosis of the amnion may result, the bacilli passing into the amniotic fluid. The fetus, by aspiration of the fluid, is thus infected with resulting primary tuberculosis of the lungs. Such cases may be considered bronchogenous infection. However, of the cases of intra-uterine infection reported, none survived a year. Nearly all succumbed within a few weeks. Most were stillborn.

However, while all this is possible, it appears to be a rare phenomenon. According to Löwenstein[2] only about 30 authentic cases of tuberculosis of the placenta have been reported in the literature.

Congenital Tuberculosis.—The rarity of intra-uterine infection is also evident when we bear in mind that cases of congenital tuberculosis are comparatively rare. It has been repeatedly stated that it is very frequent in cattle. But, according to Lubarsch,[3] there have altogether been reported only 230 cases of congenital tuberculosis in calves under three days of age, of which 215 may be considered as due to placental transmission. In humans it appears even more rarely, and of those reported, only a small proportion can be considered real instances of

[1] Ziegler's Beitr., **9**, 428; München. med. Wchnschr., 1904, p. 1576.
[2] Vorlesungen über Tuberkulose, Jena, 1920, p. 175.
[3] Ztschr. f. Aerztliche Fortbildung, 1918, **15**, 144.

congenital tuberculous disease. The first undoubted human case was reported by Schmorl and Birch-Hirschfeld.[1] The mother died from acute general miliary tuberculosis in the seventh month of pregnancy. The placenta appeared normal macroscopically, but microscopically tuberculous changes were found, and tubercle bacilli were demonstrated in the blood from the umbilical vein. Apparently the mother infected the fetus shortly before death. Londe[2] was the first to investigate the offspring of tuberculous mothers by inoculation tests, and he obtained positive results in some cases—guinea-pigs were infected when inoculated with the placental tissue, the fetal blood, and other organs. The placenta was found the most virulent tissue. Warthin and Cowie[3] reported several cases in this country, but even they warn that "intra-uterine transmission of tuberculosis is possible, but extremely rare, and needs to be supported by further research before it can be taken as final." Martha Wollstein[4] described a case in which the mother died six days after confinement, and the child died nineteen days after birth. The placenta showed advanced tuberculous changes, and the infant showed miliary tuberculosis of the lungs, spleen, kidneys, and mesentery. It is, however, noteworthy that tuberculosis of the placenta, which is more common, may not affect the fetus. Thus, A. S. Warthin[5] and Carl Vernon Weller[6] have reported cases of placental tuberculosis, and still the infants thrived for months after delivery.[7]

Another point is that it is rare that tuberculous changes should be found macroscopically in newborn tuberculous infants in whom tubercle bacilli are demonstrated microscopically and by inoculation tests. This form of congenital tuberculosis has been named by Honl *Status bacillaris* with a view of distinguishing it from true congenital tuberculosis with structural changes of a tuberculous nature; in the former, no macroscopic nor microscopic changes are found; while in the latter they are found, though both are capable of infecting when the tissues are injected into animals.

Of the cases which have been reported as congenital tuberculosis, very few are accepted as such by careful critics. In most, it has been shown the evidence is against their being really instances in which intra-uterine infection took place. Thus Péhu and Chalier[8] found only 51 authentic cases on record in medical literature. It may be added that most of the cases were not conclusively proved. Römer[9] knows of but 30 cases, and some of them may be said to be reliable only "in all probabilities." Péhu and Chalier believe that in these cases infection usually takes place at the end of pregnancy when the placental

[1] Ziegler's Beitr., 1891, **9**, 428. [2] Rev. de la tuberculose, 1893, **1**, 125.
[3] Jour. Infect. Dis., 1904, **1**, 140; Ibid., **4**, 347.
[4] Arch. Pediat., 1905, **22**, 321.
[5] Arch. Intern. Med., 1916, **17**, 509. [6] Jour. Am. Med. Assn., 1913, **61**, 1951.
[7] A good review of the literature of congenital tuberculosis may be found in F. Parkes-Weber's paper in the British Jour. Children's Dis., 1916, **13**, pp. 321 and 359.
[8] Arch. de méd. des enfants, 1914, **17**, 721. [9] Loc. cit., p. 276.

circulation is established and results from a bacteremia which is usually a terminal event. They should therefore be regarded as examples of *transplacental heredocontagion*, and not of direct heredity.

It is thus shown that, theoretically, placental transmission of tuberculosis is possible. But available facts combine to prove that it is exceedingly rare among human beings. Indeed, when compared *with the enormous number of infections after birth, the few recorded instances of congenital tuberculosis sink into insignificance.* After all, when it does occur at all, it is from mothers who are in the far-advanced stages of phthisis, or who have tuberculous disease of the genito-urinary system. Such women conceive only rarely. It is a fact worthy of note in this connection that numerous examinations of stillborn fetuses from phthisical mothers have not revealed any traces of tuberculous infection; even inoculation experiments have failed in most cases.

Among cattle congenital tuberculosis appears to be more frequent than among humans. Still, the application of the well-known Bang system has shown that, even here, it is exceedingly rare. In the United States, Harlow Brooks[1] has shown that when calves are removed from their tuberculous mothers immediately after birth, they do not develop the disease.

Handicap of the First-born.—Among the various constitutional and congenital factors in the etiology of tuberculosis there is also to be mentioned that the first-born in a family is most likely to develop the disease. As is well known, in many countries the first-born has certain prerogatives, especially among the so-called noble classes. But statistical evidence gathered by geneticists tends to show that their vitality is of inferior quality when compared with those who succeeded in the order of birth: They weigh less, are more frequently stillborn, abortions being more frequent among newly married women, and of those who come into the world alive a much larger proportion succumb during the first year of life than of the later-born children. Karl Pearson,[2] Sören Hansen,[3] and others have shown statistically that inferior physique, feeble-mindedness, epilepsy, and especially tuberculosis are more likely to occur in first-born, then in the second- and third-born, and least in those who are the fifth, sixth, etc., in a family.

Pearson showed that whereas the average incidence of tuberculosis would be 67 if the patient's number in the family was of no etiological moment, the actual incidence for the first-born was 113; among the second-born the ratio of the actual to the expected being 79 to 64. In a study of 3522 tuberculous patients at Copenhagen, Hansen found that there were 386, or about 64 per cent more first-born than there should be if the birth number was of no consequence. Among 2113 patients from the Boserup Sanatorium he found that the second- and

[1] Am. Jour. Med. Sci., 1914, **147**, 718; Tr. Soc. Exper. Med. and Biol., 1914, **11**, 50.
[2] A First Study of the Statistics of Pulmonary Tuberculosis, London, 1907.
[3] Eugenics Review, 1913, **5**, 252.

third-born are also more liable to develop phthisis, as can be seen from the following table:

Number in family.	Men.		Women.	
	Calculated.	Actual.	Calculated.	Actual.
1	328	551	274	437
2	311	389	261	324
3	282	313	238	255

It may be suggested that the first-born of tuberculous parentage being older, have been exposed to the risks of infection for a longer time and they thus swell the number of tuberculous patients. But H. Kjerrulf found among school children the number giving positive skin reactions to tuberculin is not larger among the first-born than among the later-born.

Those interested in problems of eugenics point to the fact that, inasmuch as the tendencies to small families have of late become very pronounced in certain countries, the inferior stamina of the first-born are bound to become of greater danger as regards the prevalence of tuberculosis and other physical and mental defects. However, it appears that while in France, because of the low birth-rate, the proportion of first-born is larger than in other countries, the tuberculosis mortality is very high, this does not hold for other countries. It is clear that the tuberculosis mortality has been declining with the decline in the birth-rates in nearly all civilized countries. In countries with exceedingly high birth-rates, like Russia, Hungary, etc., the mortality from this disease has been extremely high.

Clinical Aspects of Heredity.—Many authors have observed certain clinical phenomena which cannot be explained otherwise than by heredity, either of the disease, or of a predisposition to it. Brehmer, and after him several other writers, found that in many cases the onset of the disease occurs at the same age in parents and children. Piéry found that in many families the children mostly succumb before attaining the age of sixteen. While many cases can be cited in substantiation of these observations, it appears that, so far, a sufficient number have not been collated to prove their significance conclusively. The familial occurrence of the disease can be explained by infection just as by hereditary transmission, as has already been stated.

Of greater moment is the inheritance of the *locus minoris resistentiæ*, which Brehmer described long ago, and Turban,[1] Baldwin,[2] Möller, Kuthy,[3] and others have confirmed it. It appears that when pulmonary tuberculosis occurs in parents and children, the chances are immense that the same side of the chest should be affected in each case. This family resemblance in phthisis has been found in about 75 per cent of cases. In my own experience I also observed that in about two-thirds of cases the side affected was the same in the several affected members

[1] Ztschr. f. Tuberk., 1900, **1**, 30.
[2] Yale Med. Jour., 1902, p. 215.
[3] Ztschr. f. Tuberk., 1913, **20**, 38.

of the family. Möller[1] points out that when a child suffers from a tuberculous lesion of some bone, the chances are that when its brother or sister develops tuberculosis it will also be a disease of bone, and not of the soft tissues. These facts are explained by the assumption that some organs or tissues in the body lack powers of resistance, and that this defect is transmitted by heredity. This will be discussed again when speaking of the thoracic anomalies and their relation to phthisiogenesis. Meanwhile it may be stated that these problems have not received the careful study they deserve.

Disturbances in the Metabolism as Predisposing Factors.—In the search for the factors predisposing to phthisis many have looked into the metabolism of the body, stating that tuberculous infection is harmless in the vast majority of persons, so long as the metabolic processes are normal; only when certain disturbances occur in this regard can phthisis develop. It is, however, a fact that in the enormous literature on the subject of tuberculosis, we cannot find an exhaustive study of the metabolism of persons affected with the disease, and hardly anything about the metabolism in the so-called pretuberculous stage.

Several authors have maintained that an excessive excretion of calcium in the urine can be found in all cases of phthisis long before the onset of the disease. In this country, Croftan,[2] John F. Russell, and, more recently, John O. Halverson, Henry K. Mohler and Olaf Bergeim[3] have made some studies along these lines. The last-named investigators have found that the calcium content of the blood of patients with advancing and convalescing tuberculosis revealed that in incipient cases in which the patients, who were on a high milk diet, showed marked improvement, the values for calcium in the serum were normal and fairly constant. In advanced cases the variations obtained were greater (some rather high and some rather low values being obtained), and improving patients showed on the average slightly higher values than the unimproved. No marked deviations from the normal, however, were observed in the calcium content of the serum of patients in various stages of pulmonary tuberculosis. It is the opinion of these investigators that the failure of the body to deposit lime around the tuberculous areas is to be ascribed not to a deficiency in blood calcium, but rather to an inability of the cells of the tuberculous area to utilize available calcium.

Several French *savants*, notably Robin, Binet, etc., have found that in the pretuberculous stage there is a pronounced excess in the excretion of inorganic salts in the urine, notably those of lime and magnesia. The result is that the blood, bones, and lung tissues show a distinct lack in these mineral salts. Gaube found that the descendants of phthisical subjects excrete on the average more calcium and magnesia

[1] Lehrbuch d. Lungentuberkulose, Berlin, 1910, p. 30.
[2] Sixth Intern. Cong. Tuberc., 1908, **1**, 275.
[3] Jour. Am. Med. Assn., 1917, **68**, 1309.

than those of healthy stock. Robin sees in this lime and magnesia starvation an excessive amount of self-combustion, and he considers this anomaly in the metabolism the main element in the preparation of the soil prone to tuberculosis, whatever the remote cause may be—heredity, alcoholism, malnutrition, overwork, etc. Infection alone is insufficient to produce phthisis, as is evident from the fact that most people infected with tubercle bacilli escape the disease. It is only when the soil is prepared by the dissimilation and emaciation, by pretuberculous decay, as Robin calls it, that phthisis may develop. The gravity of the pulmonary lesion goes hand-in-hand with the degree of lime starvation, demineralization and emaciation of the body. According to these writers, phthisis is preventable. Demineralization of the body must be sought and, when discovered, prevented by the administration of remedies tending to replace the lime and magnesium which are being eliminated from the body excessively.

These and other findings about the metabolism in phthisis have not been confirmed by all who have made careful studies along these lines. It appears that in the vast majority of consumptives the metabolism is quite normal so long as there is no high fever. The occasional lapses in the metabolism are explained by the usual causes of morbid phenomena observed in other diseases characterized by fever, emaciation, debility, etc. This has been confirmed by the studies of McCann and Barr,[1] who found that the basal metabolism of tuberculous patients may be normal or slightly above that of normal men of the same size. With the increase in the temperature there may occur an increase in the basal metabolism, but it is not large. The basal heat production in tuberculosis may be less than the normal for the same patient in health. In other words, the loss in weight may be accompanied by a reduction in metabolism which more than compensates for the tendency to increase caused by the disease. However, the basal metabolism in the pretuberculous stage, *i. e.*, in those in whom tuberculosis has not yet been developed fully in the clinical sense, has not been studied as yet. We do not know whether any changes in this direction predisposes to the evolution of tuberculous disease. At any rate, this subject has not been studied sufficiently to permit making generalizations.

Endocrine Disturbances.—Recent studies of endocrine disfunctions have shown that changes in the structure and functions of the internal secretory glands are very frequently encountered in tuberculous patients, though hardly any etiological relationship has been established. Considering that tuberculosis is so widely prevalent, it is to be expected that some should present evidence of glandular disturbances. However, it appears that certain glandular conditions favor the development of tuberculous disease, while others prevent it more or less. While studies on the etiological relationship of the endocrines

[1] Arch. Intern. Med., 1920, **26**, 663.

to tuberculous disease have not been as thorough and as abundant as appears desirable, still some facts are already available which are very suggestive.

As will be shown later on (see Chapter XXX), excessive secretion of the thyroid gland protects, in a measure, against the development of active and progressive tuberculosis. It appears that hyperthyroid individuals only rarely suffer from active phthisis, and when symptoms of this disease present themselves, the pulmonary lesion is mild, and manifests strong tendencies to cicatrization. This clinical fact was already noted by Hamburger as far back as 1853, and in 1887 S. Solis Cohen pointed out that a large thyroid is characteristic of immune members of tuberculous families. Morin[1] observed that in familial thyroid disease, the members of the family that had large thyroids with hyperfunction of the gland did not develop tuberculosis, while 25 per cent of 348 cases with atrophy of the thyroid became tuberculous. "Atrophy, or defective development of the thyroid," says Sajous,[2] "is commonly observed in persons predisposed to tuberculosis and other infections. Indeed, the prominence of Adam's apple in such subjects is due to the fact that a flat thyroid fails to fill sufficiently the infrathyroid portion of the neck to preserve its normal shape. (Emaciation, however, has a great deal to do with it.) Such a diminutive thyroid may be due to deficient antenatal or postnatal development, but in many instances it is traceable to infectious disease." He attributes the therapeutic value of iodine in tuberculosis to the iodine content of the thyroid gland. Bauer, Turban, Muralt, Sergent, Rénon, and others have confirmed these observations. In this connection it is important to mention that hypothyroid individuals suffer from tuberculosis more often than others, as will be shown in another part of this book.

The etiological relationship between adrenal function and tuberculous disease is even more evident. The low blood-pressure in the pretuberculous stage, the myasthenia, weakness, and especially the brown pigmentation of the skin, all of which are common symptoms of active phthisis, speak for disfunction of the adrenal glands. Sajous says that lesions of the adrenal, clinical or experimental, increase materially the vulnerability to infection. "In tuberculosis, in fact, adrenal stigmata are very common, 24 per cent according to Lafitte and Moncanny, and 20 per cent according to Laignel-Lavastine of cases taken at random having shown more or less bronzing —a sign of advanced lesions of the adrenal tissues." Bauer suggests that outdoor life, which has been found efficacious in phthisiotherapy, usually produces bronzing of the skin, which, according to some authors, is an indication of functional activity of the accessory chromaffin tissue, counteracting the tuberculous toxemia. Sergent, Gerald Webb, and many others have used adrenal therapy in certain cases

[1] Rev. Méd. de la Suisse Romande, 1895, **15**, 241.
[2] New York Med. Jour., 1917, **106**, 389.

of tuberculosis with good results. Experimentally Gerald Webb[1] and his co-workers have found that tuberculous infection is followed by enlargement of the thyroid and adrenals, probably in response to a demand for increased function. It must, however, be mentioned in this connection that hypofunction of the adrenals, observed in many cases of phthisis, is usually due to tuberculous lesions of these glands, and may be considered a result of tuberculous disease more than an etiological factor. On the other hand, myasthenia, vascular hypotension, etc., are early symptoms, so early that some writers have been tempted to call them "pretuberculous."

The etiological relationship between gonadal function and tuberculosis deserves careful study. Clinical observations appear to favor the view that hypofunction of the sexual glands has a salutary influence on existing tuberculous disease. Bauer[2] speaks of this relationship as beyond any doubt. He has never observed a case of tuberculosis in a eunuch. Further on we will show that when symptoms of eunuchism develop in persons with tuberculosis of the testicles, tuberculosis of the lungs is uncommon. Warnekros has observed this fact in castrated women, and Mauthner in castrated guinea-pigs. A very large proportion of women who develop pulmonary tuberculosis during or soon after the onset of the menopause suffer but little from the pulmonary lesion which tends to sclerosis; fibroid phthisis in general occurs mainly in persons over forty years of age, when the functions of the sexual glands are rather low. On the other hand, during the period of puberty, when the sexual glands are at the height of functional activity, tuberculosis is apt to run a progressive course.

We know very little about the etiological relationship of disfunction of other endocrines to tuberculosis.

Predisposition of the Lungs.—More than nine-tenths of all tuberculous disease in humans is found in the lungs; animal experimentation also shows that these organs are more likely to suffer as a result of tuberculous infection than any other structure. No matter how experimental infection is accomplished, by subcutaneous or intraperitoneal inoculation, by intravenous injection, by inhalation or ingestion of the bacilli, there occurs almost invariably a lung lesion sooner or later, irrespective of the other organs that may be affected by tubercle.

Some, notably Bartel,[3] believe that this is due to the well-known defective development of the pulmonary lymphatic system. The lymphatic structures are excellent weapons against all infections, and, possessing poorer lymphatics, the lungs are thus more liable to tuberculous changes after the specific virus is brought into them with the inhaled air, or the blood stream. Those who favor this viewpoint to physiological anthracosis of the bronchopulmonary lymphatics, met with in nearly all adult city dwellers, as reducing the functional

[1] Am. Rev. Tuberc., 1921, **5**, 266.
[2] Med. Klinik, 1921, **17**, 1045.
[3] Pathogenese der Tuberkulose, Berlin, 1918.

capacity of these structures. However, clinical experience shows clearly that anthracosis does not predispose to tuberculosis in the vast majority of cases. In fact, coal miners, whose pulmonary lymphatics are glutted with soot, are less liable to tuberculosis than others (see p. 132).

The anomalous circulation of blood in the lungs has been considered by some as responsible for the frequency of tuberculosis in them. It is mentioned in this connection that they are not supplied sufficiently with arterial blood. As will be shown later on, oligemia of the lungs, in fact, predisposes to pulmonary tuberculosis, as is the case with pulmonary stenosis (see p. 127); on the other hand, in pathological conditions involving pulmonary hyperemia, as in mitral stenosis, tuberculosis is exceptional.

Certain investigators have found lung tissue an excellent culture medium for the growth of tubercle bacilli. Neumann and Wittegenstein,[1] A. E. Porter,[2] and others, have attempted to culture tubercle bacilli on various organs and found that while in most tissues the virus loses its virulence, in lung tissue it retains it. Porter found that liver, thymus, and lymphatic gland were strongly bactericidal, pancreatic extract the most powerful, while lung tissue was the least bactericidal. It has also been suggested that while in most other organs there is, as a rule, to be found a lypolytic ferment, the lung lacks it. But Sieber and Dzierzgowski[3] have shown that this assumption has no foundation in fact. In this connection it is interesting that Abderhalden speaks of demonstrable ferments in the pulmonary alveoli which, he states, have their origin in the leukocytes which migrate into them in large numbers to fight invading pathogenic microörganisms. Another suggestion as to why tubercle bacilli thrive in lung tissue is one propounded by M. Weisz[4] to the effect that it lacks in oxidizing ferments, oxydases. The connective tissue in the lungs is known to be deficient in nuclei which, in all probabilities, is an adaptation to the oxygen-carrying function of the lung. In other words, the predisposition of the lung to tubercle is alleged to be the result of the inferior oxidizing power of its tissues. Still another suggestion has been made: Tubercle bacilli in the lymphatic system enter the veins, and are carried by the blood first through the right heart. The lung is the first viscus they strike.

Because of the narrow flat chest seen in many tuberculous patients we are apt to think that their lungs are smaller than normal. But actual investigations have shown that Rokitansky was right when he stated that the lungs in the tuberculous are relatively more voluminous than in others. It appears that the narrowness and flatness of the chest is compensated by its excessive length. In addition there is found

[1] Beitr. z. Klinik. d. Tuberk., 1909, **12**, 145.
[2] Jour. Hygiene, 1917, **16**, 55.
[3] Ztschr. f. physiol. Chemie, 1909, **52**, 254.
[4] München. med. Wchnschr., 1912, **53**, 697.

a small heart, defective structure of the arterial walls, and a small abdominal cavity with correspondingly small abdominal viscera. These peculiarities have been observed by many pathologists, and Brehmer insisted that the chief characteristic of the phthisical chest consists in a disproportion between the voluminous lungs and the small heart. The abnormally large lung, with its correspondingly large capillary network, is insufficiently nourished by the amount of blood which the small heart and hypoplastic bloodvessels can send forth, and is thus more exposed to attack by the tubercle bacilli which are brought there by the blood or air stream. The comparative immunity to tuberculosis of individuals who present stigmata of lymphatism, which is discussed elsewhere in this book, is in agreement with the hypothesis that an abnormal and defective circulation of the blood in the lungs is responsible for its excessive predisposition to tuberculosis. The fact that the terminal bronchioles are not supplied with cilia is also suggestive. Tubercle bacilli, reaching there with the inhaled air, may settle within the air sacks, and are then carried by the lymph vessels farther on, until arrested in the lymphatic glands.

Predisposition of the Pulmonary Apices.—It is, however, important to recall that the entire lung is not subject to tuberculous changes, excepting in acute miliary tuberculosis and the terminal stages of chronic phthisis. The point of election for tuberculous lesions is the uppermost part of the lung, the apex, which is in itself an argument against the inhalation theory of the disease. *A priori* we should expect that gravity should rather carry tubercle-laden air downward, into the lower lobes of the lungs.

Various hypotheses have been suggested to account for the apical localization of phthisis. Some have maintained that it is due to the lesser functional activity of the upper part of the chest which expands but slightly even in the female, in whom the pectoral region, and not the real pulmonary apex, expands during ordinary breathing. Tendeloo[1] showed that expansion of the air vesicles differs in the various parts of the lungs; it is least in the paravertebral and the uppermost parts, and the maximum occurs in the lower and anterior-lateral parts. Renewal of air is proportional to the expansion of the air vesicles. For this reason, tubercle bacilli brought in with the inspired air, as well as with the lymph stream, have more chance of remaining in the apices, thus favoring implantation at that point. In the adult the uppermost portion of the lung extends for about one to one-and-a-half inches above the first rib, and that portion is only covered by soft tissues; it is thus deprived of the inspiratory pull of the bony framework of the chest, is more exposed to the external atmospheric pressure, and therefore any impediment to the exit of the air current, due to any cause, also impedes the proper expansion of the apex, and atelectasis of this portion of the lung is favored, retaining germ-laden air. While

[1] Studien über die Ursachen der Lungenkrankheiten, Wiesbaden, 1902.

strong respiratory movement of the chest will easily ventilate the lower lobes of the lungs, the ventilation of the apices is rather defective for the above-mentioned reasons.

It appears, however, that this mechanical explanation does not clear up the problem. We should expect that in pneumokoniosis the dust would be retained mainly in the apices, which is not the case. In fact, most authorities agree that in pneumokoniosis the upper parts of the lungs are comparatively free from deposited dust, while the lower and posterior parts may be filled with the aspirated material.

Other writers attribute the predisposition of the apices to their defective circulation. They have not the rich blood supply of the lower lobes. In addition, according to Tendeloo, the apices have a defective lymph circulation. While all other large bronchi proceed from the main bronchi in a straight direction, or at an obtuse angle, so that the air can pass straight ahead in either direction during inspiration and expiration, the apical bronchus forms an almost right angle, and the movement of the inspired and expired air is impeded. Difficulty in the proper ventilation of the apical portions of the lungs is thus created. Rindfleisch maintained that the bronchial secretions in the apices are thicker and more viscid than in other tubes, because of the relative dryness of the apices owing to paucity of the blood, the effects of gravity.

More recently Cobb[1] suggested, and brought considerable proof in substantiation, that the apical localization of phthisis is due to defective glandular supply of that part of the lung. Tubercle bacilli, having no selective power for any particular portion of lung tissue, will grow as rapidly in one portion as in another, once they are arrested and gain a foothold. In man, that portion of the parietal pleura lying contiguous to the apex of the lung is drained by a vast network of lymph vessels that empty into the deep cervical chain. Nowhere else in the human thorax is there a decided counter lymph current; and, granting that there is this counter current, it could account for an area of lymph stasis in the apex of the lung. This is in agreement with the views of Tendeloo, and especially Bartel, mentioned above.

Freund's Theory of Stenosis of the Upper Thoracic Aperture.—About sixty years ago Freund[2] pointed out that stenosis of the bony thorax is very frequently encountered in consumptives, but his observations were neglected and soon forgotten, to be taken up again by himself, Hart and Harras,[3] and others. Bacmeister's[4] experimental investigations have finally given great plausibility to Freund's theory.

The deformity of the upper thoracic girdle, which may be congenital or acquired, consists mainly in an ossification of the first costal cartilage and a shortening of the first rib which exerts pressure upon the lung

[1] Jour. Am. Med. Assn., 1918, **70**, 1511.
[2] Beitr. z. Histologie d. Rippenknörpel, Breslau, 1858.
[3] Der Thorax phthisicus, Stuttgart, 1908.
[4] Die Entstehung der menschlichen Lungenphthise, Berlin, 1914.

apex which it surrounds, thus obstructing the circulation of the blood and lymph, preventing the removal of any foreign body—the tubercle bacilli—that may be brought there by the blood or the inspired air, and favoring its localization at this point. Shortening of the first costal cartilage also involves an excessive inclination of the upper thoracic aperture toward the spinal column. The sternum lies too deeply, the ribs run slantingly downward, the shoulders hang low and forward, the scapulæ protrude like wings, and the result is the phthisical chest of the classical authors.

Freund, Hart, and Harras have studied the tuberculous thorax on the autopsy table and in the living with the aid of roentgenography, and have found that stenosis of the upper aperture is very frequent. The abnormal shortening of the first rib makes the transverse diameter short, converting the human thorax into one like that of the lower animals, and to a certain extent infantile, as is shown in Fig. 15. The narrowing usually occurs at the latero-posterior bulging, exactly where

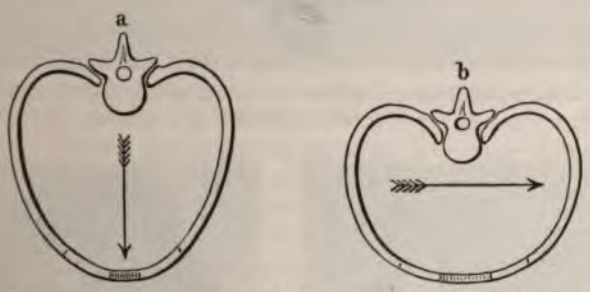


FIG. 15.—Diagrammatic representation of the upper aperture of the thorax: *a*, the primary form (animals, primitive human form); *b*, secondary form (adult man). (After Wiedersheim.)

the apices of the lung are surrounded by the first rib, which under these conditions compresses the pulmonary tissues beneath. This deformity may occur unilaterally or bilaterally, but the end-result is always the same—narrowing and rigidity of the upper thoracic girdle with resulting compression of the lung.

Independent of Freund, Schmorl[1] found a groove about 2 cm. below the highest point of the apex of the lung. This groove is very frequently encountered in newborn infants, but in them it can be obliterated when the lung is inflated. During adolescence it disappears in persons with normal chest walls. In most persons in whom it persisted Schmorl found tuberculous lesions beneath the point which was pressed upon by the shortened rib (Figs. 17 and 18).

These observations have been confirmed by Birch-Hirschfeld[2] from another point of view. While searching for the initial lesion of tuberculosis in cadavers dead from other diseases, he found that phthisis

[1] München. med. Wchnschr., 1902, **48**, 1995.
[2] Deutsch. Arch. f. klin. Med., 1899, **64**, 58.

begins in the walls of a bronchus of the third to the fifth order, and ascribed it to certain pressure exerted on these tubes, preventing the exit of air and secretions. This bronchiole, which Clifford Allbutt

FIG. 16.—Upper aperture of the thorax: *A*, normal on left side; *B*, narrowed at the right. (Freund.)

FIG. 17.—Right lung. (His's model.) The indentations made by the ribs are shown. The first groove is the indentation made by the first rib and is known as Schmorl's groove.

FIG. 18.—Left lung. The groove of the first rib is shallower than in the right lung.

calls "Hirschfeld's bronchiole," from its position and nature favors that secretions, instead of clearing themselves automatically, will stagnate more or less if pressed upon to a greater or lesser degree by the first rib, located as it is on the apex, leading spirally against the action of gravitation upward, outward, and backward.

Finally, Bacmeister's[1] investigations have apparently confirmed these anatomical, pathological, and clinical findings. He surrounded young and growing rabbits with a wire loop at the first costal ring. thus causing stenosis of the upper aperture of the bony thorax. The pulmonary apex was thus compressed, and a groove was indented in the lung beneath the wire loop corresponding to the one observed by Schmorl in human consumptives. Infecting these animals, he produced isolated and localized pulmonary tuberculosis, while in normal animals, used as controls, infection produced miliary tuberculosis, but never localized tuberculosis of an apex. In this manner he could produce local tuberculous lesions on either side of the chest, or bilaterally.

Considerable clinical evidence has been brought in support of this theory. In children the upper aperture of the thorax is very elastic, and therefore apical phthisis is exceedingly rare; when infected, the tracheobronchial glands are affected, or general miliary tuberculosis is the result. During the period of puberty, when the spinal column grows and raises the upper thoracic girdle, permitting the first rib to exert pressure on the pulmonary apex, typical phthisis may occur. The largest number of cases of active tuberculosis of the lung, though not the largest number of deaths due to this cause, occur between fifteen and thirty years; between thirty and forty the proportion diminishes, and between forty and sixty there again occur a large number of cases. Hart explains these phenomena in this manner: During puberty and soon thereafter any congenital or acquired shortening of the first rib becomes dangerous to the individual because the growing apex of the lung finds itself enclosed in the small and rigid thoracic cavity, which does not grow in the same proportion as the lung, and the shortened first rib compresses it, thus favoring tuberculous degeneration. After forty, when ossification of the costal cartilage is, to a certain extent, normal, conditions are again favorable for the development of phthisis.

While several authors have confirmed these findings by Freund and Hart, it appears that careful observers have looked for stenosis in the upper thoracic aperture while making autopsies, or roentgenographing tuberculous subjects, and could not find it in an unusually large proportion. Schulze, Smith, Neumann,[2] and finally Wenckenbach, have all obtained negative results. Roentgenography of 273 tuberculous individuals by Neumann has not shown abnormal shortness, or excessive ossification of the first rib and cartilage. Among 238 cases

[1] Mitt. a. d. Grenzgeb. d. Med. u. Chir., 1913, **26**, 630.
[2] Beitr. z. Klinik. d. Tuberkulose, 1919, **40**, 1.

of tuberculosis, Wenckenbach[1] found that 35.7 per cent showed no trace of ossification of the first cartilage, 25.5 per cent a slight ossification which is normal in healthy individuals over thirty years of age. It is thus seen that in 61.75 per cent of tuberculous subjects this ossification cannot be credited with etiological relationship to the tuberculous process in the lungs, and that only in 17.2 per cent has this anomaly been found to exist. In younger individuals it is even less frequently found. Thus, among 172 cases of tuberculosis in persons between fifteen and thirty years of age, it was found only in 1.5 per cent, while in 86.3 per cent no trace of the mentioned anomaly could be discovered.

Arthur Keith,[2] Stiller,[3] and other authors are inclined to look upon this deformity of the thoracic girdle rather as a result of tuberculosis than a cause of it. Pottenger[4] points out that the muscle change described by Freund as hypertrophic and due to overwork, caused by the muscle pulling against an ankylosed rib, is more likely a contraction of the muscle caused by the inflammation within the lung reflexly through the spinal cord.

Constitutional Inferiority.—Many writers have maintained that predisposition to tuberculosis is not specific, due to anomalous structure or function of a given organ, but that it is general. They claim that the majority of tuberculous patients present signs of constitutional inferiority; others allege that "stigmata of degeneration," in the sense given this expression by Lombroso, are preponderating.

The application of the term *dégénéré* to tuberculous individuals was first made by the noted French clinician Andral, and Féré stated that the phthisical present very frequently stigmata of degeneration. Then a Polish writer, Polansky,[5] made some extensive, though not very astute, observations along these lines, while making autopsies on 854 tuberculous subjects. Among the numerous anomalies and stigmata which he said were common among these subjects are concavity of the region of the small fontanelle, persistence of the interfrontal suture to an advanced age, frequent occurrence of Wormian bones, abnormal length of the transverse processes of the lower cervical vertebræ, bifid spinous processes in the lower dorsal and upper lumbar vertebræ, etc.; also bifurcation of the xiphoid cartilage, or its perforation. He also claims that certain anomalies in the viscera are excessively found, such as hypoplasia of the cardiac muscle, lack of the middle lobe of the right lung, thinness and narrowness of the wall of the aorta, bifurcation of the aorta as high as the second or third lumbar vertebra, lobulation of the kidneys and liver, abnormal length of the right lobe of the liver, double ureters, accessory spleens, fetal length of the stomach, abnor-

[1] Wien. klin. Wchnschr., 1918, **31**, 379.
[2] Further Advances in Physiology, 1909.
[3] Berl. klin. Wchnschr., 1912, **49**, 97.
[4] Muscle Spasm and Degeneration, St. Louis, 1911.
[5] Ztschr. f. Tuberk., 1904, **6**, 140.

mally long colon, mesocolon and appendix, Meckel's diverticulum, patency of the hernial canals, etc. Excessively long extremities, and heterosexual distribution of the hair on the body are other anomalies mentioned by this writer.

Many other authors have found signs of constitutional inferiority among the tuberculous. A Japanese writer, Iwai[1] found polymastia and supernumerary nipples; Rosolimo[2] absence of the lobule, the so-called "jug-handle" ear; and Holeman[3] says that he observed stigmata of degeneration; "the most common, as well as the most striking and easily observed, are various malformations of the pinna; next to these, ill-formed palates and gross facial asymmetries abound." W. C. Rivers[4] published a book showing that certain atavistic tendencies are found in a large proportion of consumptives, notably ichthyosis, mancinism, squint, etc.

The *asthenic constitution*, which has been described in detail by Stiller, is said by some authors to be predisposing to tuberculous disease. People of this type have long chests and necks, winged scapulæ, protruding clavicles and second rib, and very defective and hypotonic musculature, involving ptosis of the abdominal viscera, etc. In this country, Joel E. Goldtwait[5] has arrived at the conclusion that "the congenital viscero-ptotic, the carnivorous, the hyper-onto-morph" is very much subject to tuberculosis.

The complexion of the individual also has been considered in this regard. Some 2000 years ago Hippocrates said that blondes are more likely to develop consumption than brunettes. Recently others have maintained the same. Red hair is especially said to be predisposing by Rivers,[6] Bauer,[7] and many others. Schmidt states that "hair disharmony,"—bright red mustache, with dark drown hair on the head—is found in excessive proportion in patients with tuberculous peritonitis.

These anomalies of pigmentation are due to ethnic inter-mixtures. We have seen that racial peculiarities have very little to do with tuberculous infection, excepting in so far as to the length of time a given ethnic unit has been exposed to tubercle bacilli (see p. 73). But many writers have maintained that consanguineous marriages are predisposing to tuberculosis in the offspring. However, a more recent author, Herman Lundborg,[8] found that races that avoid intermixture are more or less immune; and wherever racial miscagenation is intense the tuberculosis morbidity and mortality are high. This does not hold

[1] Lancet, 1907, **2**, 958.
[2] Wien. klin. Wchnschr., 1908, **31**, 790.
[3] Med. Record, 1915, **88**, 1037.
[4] Three Clinical Studies in Tuberculous Predisposition, London, 1917; Lancet, 1921, p. 319.
[5] Boston Med. and Surg. Jour., 1916, **175**, 88.
[6] British Jour. Children's Dis., 1920, **17**, 59.
[7] Konstitutionelle Disposition zu inneren Krankheiten, Berlin, 1917, p. 54.
[8] Swenska Läkarsällskapets Handlingar, 1920, **46**, 73; abstr. in Zentralbl. f. Tuberkuloseforschung, 1921, **15**, 338.

good for the white population of the United States nor will those who correlate anthropological with demographic data anywhere in the world agree with this view.

Other anatomical anomalies are those pertaining to the structure of the lymphatic system. Virchow, and later, Cornet, stated that certain individual variations in the lymphatic system render infection and the subsequent evolution of tuberculous disease easier. Geddes[1] also maintains that the venous and the lymphatic systems are of defective development in the tuberculous.

Clinicians, who care for and observe thousands of cases of tuberculosis have not noted that "stigmata of degeneration" are more common among them than among others. Considering that tuberculosis is extremely widespread, affecting nearly one-tenth of humanity, it is clear that among tuberculous patients there are many who present stigmata of constitutional inferiority. But it cannot be truthfully said that they preponderate. Indeed, it appears that when perfect specimens of physical development are affected, the disease, as a rule, pursues a very malignant course.

PREEXISTING DISEASES.

Disease of the Respiratory Tract.—Of the diseases which, at one time or another, have been considered predisposing to the development of pulmonary tuberculosis, those affecting the upper respiratory tract are nearly always mentioned as preparing a favorable soil for the implantation of tubercle bacilli. Thus, we occasionally meet with instances of bronchiectasis, syphilis, actinomycosis, and cancer of the lungs, and chronic pneumonia, in which tuberculosis is implanted at the site of the primary disease. There are two plausible explanations for these phenomena: In most cases it is, in all probability, an old, dormant tuberculous lesion, dating back to childhood, that is reawakened into activity by the intercurrent disease, assisted perhaps by the reduction in the vitality and resisting powers of the patient. In pneumokoniosis the foreign particles in the lung tissue are said to produce local ischemia, obstruct the lymph channels, and thus prevent absorption, or destruction, of any tubercle bacilli that may be brought in by the air stream. But, as will be shown later on (see p. 132), there are good reasons for speaking of a comparative immunity against tuberculosis by persons affected with the just-mentioned diseases. Real lobar pneumonia is hardly ever followed by pulmonary tuberculosis, and in most cases in which it has been observed, the probabilities are in favor of the primary disease being acute pneumonic phthisis which had subsided, then pursuing the course of chronic tuberculosis. Especially is this true of "apical pneumonias" and "basal tubercu-

[1] Dublin Jour. Med. Sci., 1909, **128**, 337.

losis," and many of the so-called unresolved pneumonias, when not empyemata, have been tuberculous from the start.

Pleurisy.—Of greater importance is the etiological relation of pleurisy to phthisis. Of course, the secondary pleurisies, those occurring in cases of intrathoracic neoplasms, cardiac and renal diseases, have no significance in this regard. But the forms of acute and chronic pleurisy which had formerly been considered "idiopathic," appear to be, in the vast majority of cases, of a tuberculous nature, though many are undoubtedly rheumatic.

Strictly speaking, pleurisy cannot be considered predisposing to pulmonary tuberculosis, because it is in itself a manifestation of tuberculous disease. As will be shown later on (see Chapter XXVI), it is due to tubercle bacilli, and indicates that the acute or annoying manifestations of the pathological process begin in the pleura.

Diseases of the Upper Respiratory Tract.—Because acid-fast bacilli are at times found in the tonsils, many writers have argued that this gland is one of the main channels of entry of the virus of tuberculosis, and that diseases of the rhinopharynx are predisposing to tuberculous disease of the lungs.

Clinical experience is, however, not in agreement with this view. The fact that virulent tubercle bacilli are at times found in the tonsils does not prove that they can pass further on into the deeper respiratory passages and cause local lesions in the lungs, as was already shown. Moreover, it is exceedingly rare that pulmonary tuberculosis should begin with an acute inflammatory process in the nose and throat. This point is discussed in greater detail in Chapter XXX. On the whole, it may be stated that no etiological relationship between acute and subacute inflammatory processes of the nose and throat and pulmonary tuberculosis has been found to exist.

"Colds."—Clinical observation shows clearly that exposure to cold, or chilling the body, very often precedes the onset of active symptoms of pulmonary tuberculosis. Of course, it is self-evident that chilling cannot produce tuberculous disease. But when we bear in mind that practically everybody has tubercle bacilli in some part of the respiratory tract, we can readily understand that the changes induced by exposure to cold may be instrumental in preparing a favorable soil for the reactivation of dormant tubercle bacilli. A large proportion of the patients in whom the onset of tuberculosis was marked by symptoms of pleurisy state distinctly that they had felt well until exposure while sea-bathing, or emerging from an overheated room into a cold or windy atmosphere, or being chilled by wet and cold clothing. In active cases of tuberculosis, complicating pleurisy is very frequently observed after exposure to the vicissitudes of the weather. To be sure, we are in the dark as to the exact pathological or biochemical mechanism of "catching cold." It is immaterial for our purposes whether the primary changes in the affected tissues are those of active or passive hyperemia, as has been supposed for a long time, or a reflex vaso-

constriction and ischemia, as has been found experimentally by Mudd and Grant,[1] or the more recent theory of Schade,[2] that functional disturbances in the cells of the tissues of a colloidal character, are responsible. No observant clinician has failed to be impressed with the fact that a large proportion of known tuberculous patients are distinctly harmed by exposure to cold, or chilling of the body, and many who did not consider themselves sick at all, began to show symptoms of tuberculosis soon after exposure and chilling. It is clear that in the latter class of cases, of which we meet many, tubercle bacilli were present in the thoracic organs, especially the lungs, glands, and pleura. But the chilling is here the exciting cause, reactivating dormant lesions which had given no trouble before. On this point many recent writers, notably Sticker,[3] Mohr,[4] Aufrecht,[5] Keysser,[6] Ransome,[7] and many others agree. It is unfortunate that, absorbed in bacteriology, we are apt to underestimate the predisposing, and often the exciting element of exposure to the vicissitudes of the weather in the etiology of tuberculous disease of the lungs.

There is one point to be remembered by physicians in this connection. When reactivating dormant tuberculosis of the lungs, exposure does not produce symptoms of the "common cold" such as those of coryza, rhinopharyngitis, tonsillitis, etc. The symptoms following exposure in these cases are those of incipient tuberculosis, such as cough, fever, etc., and when the pleura is involved, which is very frequently the case, symptoms of pleurisy are at once manifest.

Chronic Bronchial and Pulmonary Diseases.—Clinical experience has shown conclusively that chronic diseases of the bronchi and lungs are not predisposing to tuberculosis. The notion of some patients, and, at times, of physicians, that a "neglected" bronchial catarrh, asthma, pulmonary emphysema, etc., is at any time liable "to turn into consumption" seems to be unfounded. To be sure, there are many patients who have coughed for months or years before they have been pronounced tuberculous, but the cough, expectoration, dyspnea, etc., were, in these cases, due to quiescent, or even active, but unrecognized, tuberculosis. What is called chronic bronchitis, is in most cases secondary to cardiac or renal disease. As will be shown later on (see Chapter XXX), there is ample evidence to the effect that pulmonary emphysema and asthma are only rarely followed by tuberculosis. Exceptions that may be mentioned are the emphysematous forms of fibroid phthisis, which are due to the inhalation of certain kinds of dust, especially in miners of silica and certain metals, notably copper and tin, also in garment workers, furriers, rag-pickers, etc.

[1] Jour. Med. Research, 1919, **40**, 55.
[2] München. med. Wchnschr., 1919, **66**, 1021; 1920, **67**, 449.
[3] Erkaeltungskrankheiten und Kaelteschaden, Berlin, 1916, pp. 141, 197.
[4] In Mohr und Staehelin's Handbuch d. inn. Medizin, 1912, **4**, 759.
[5] Ztschr. f. Aerztl. Fortbild., 1917, **14**, 574.
[6] Ztschr. f. Baneologie, 1913, **6**, 421, 455.
[7] Lancet, 1888, **1**, 762.

Diseases of the Heart and Bloodvessels.—The small heart seen in a large proportion of tuberculous patients has suggested that there may be an etiological relationship between tuberculosis and the size, capacity and thickness of the walls of this organ. Many authors consider a congenital hypoplasia of the cardiac muscle a prerequisite, or at least a predisposing factor, in tuberculosis. Moreover, with an hypertrophied heart, active tuberculosis of the lungs is exceedingly rare. The reason for this phenomenon is given as follows: Conditions of the heart which produce oligemia of the lung seem to favor the development of tuberculosis, and this is perhaps best illustrated by the fact that nearly all who have congenital heart disease, pulmonary stenosis, suffer from and succumb to tuberculosis. On the other hand, diseases of the heart which produce congestion and plethora of the pulmonary bloodvessels, as is the case in mitral stenosis, are antagonistic to tuberculosis. The fact that tuberculosis is very rare in patients with mitral stenosis favors this view. The clinical aspects of this problem are discussed in Chapter XXX.

Acute Infectious Diseases.—It has repeatedly been observed that the endemic contagious diseases, like measles, scarlet fever, whooping-cough, diphtheria, etc., are often followed by phthisis, and in infants and children tuberculous bronchopneumonia is frequently a sequel of measles and whooping-cough. This heightened predisposition may be explained as depending on the general disturbance in health caused by the fever, catarrh of the respiratory passages, etc., which reduce the resisting power and produce a soil favorable for the activation of dormant foci of tubercle bacilli, or favor new infections. These diseases are accompanied to a great extent by irritation of the mucous membranes and defects in the epithelium which facilitate the entrance of the bacilli, so that infection of the respiratory passages is particularly favored. The influence of measles and whooping-cough may be purely mechanical; fits of violent cough are liable to rupture tuberculous glands in the chest.

In children tuberculous bronchopneumonia is very frequently observed to follow an attack of measles. In adults our experience had been limited till the epidemic which broke out in various camps in which United States soldiers were stationed during 1917–18. Among 5945 cases of measles in soldiers it was found that 173, or 2.91 per cent., had developed active tuberculosis. George E. Bushnell[1] is inclined to the opinion that the measles reactivated latent tuberculous foci, though he believes that it is probable that the number of really tuberculous cases was less than the above figures would indicate. Some of these cases classed as tuberculous were rather cases of unresolved pneumonia. But, on the other hand, he has no doubt that all cases of tuberculosis reactivated by measles had been detected at the first examination of these soldiers. This may be considered an experiment on a large scale

[1] Jour. Am. Med. Assn., 1918, **70**, 1823.

which tends to show the influence of measles on the incidence of tuberculosis.

That these diseases may be strong predisposing factors to tuberculous infection and the extension of existing tuberculous disease, was shown from another viewpoint. "Allergy,"[1] or the altered reactivity of the organism to tuberculin, is apparently dependent upon the fact that the body has produced antibodies which counteract the effects of tuberculous toxemia, and is diminished in intensity, or disappears altogether, during an attack of measles. We then have "anergy," which indicates that resistance to infection has diminished, just as in far-advanced phthisis for a short period before the fatal termination, in miliary tuberculosis, etc., when all defensive powers have failed. Von Pirquet has named this state "anergic," *i. e.*, non-reacting. He assumes that the measles process occupies the antibodies which are needed for the repulsion of the tubercle bacilli present in the body. During this unprotected period the tubercle bacilli can grow and pass through the necrotic walls of a caseous gland, or secondary diseases can also occur, because now the circulating tubercle bacilli can find favorable conditions where at other times they would have been destroyed. He draws an analogy between this condition and the condition favoring the progress of tuberculosis in the adult—general debility due to malnutrition, overwork, or any other condition robbing the body of its natural defences.

Influenza.—The connection between influenza and phthisis is even less clear. During the great pandemic of influenza in 1891 it was observed that the mortality was increased, and similar observations had been made before. Arthur Ransome[2] called attention to the periodic waves in the death-rate from phthisis in England and Wales, and noted faint indications of a rise in 1853, 1866, 1878 and 1890. Bulstrode, in referring to these rises in the mortality, pointed out that there was an outbreak of influenza in 1855 which might possibly account for the increase in tuberculosis at that time. But in 1866 the cotton famine accounts for it much better. During 1890–91–92, and again in 1899–1900, the mortality from phthisis increased as a concomitant to epidemics of influenza. As Newsholme[3] points out, the experience of 1917–18 was the third occurrence in recent years of this coincidence, and there can be no doubt that influenza is a most dangerous complication of pulmonary tuberculosis. But during the two years following the epidemic the tuberculosis mortality declined in England. During the epidemics of influenza in the United States in 1918–1919, I observed that those who recovered showed no tendency to develop phthisis, unless they had tuberculous lesions before the attack of influenza. As will be shown elsewhere (see Chapter XXX), when a tuberculous person is stricken with influenza, the outlook is not invariably bad.

[1] See von Pirquet, Allergie, Ergebnisse d. inn. Med. u. Kinderheilk., 1910, **5**, 459.
[2] Tr. Epidemiol. Soc., London, **24**, p. 252.
[3] Lancet, 1917, **2**, 591.

Typhoid Fever.—Typhoid fever also has been considered as predisposing to phthisis because of the rather high proportion of consumptives who give a history of having passed through an attack of it. Recently, Charles E. Woodruff[1] has discussed the subject in great detail and arrived at the conclusion that typhoid fever heads the list of predisposing causes of tuberculosis. The fact that during recent years the mortality from tuberculosis and from typhoid has been declining at almost the same rate is considered a strong argument. "The three diseases which seem to be most frequently followed by tuberculosis of the lungs—measles, whooping-cough, and typhoid—are all complicated with bronchitis."

There appears to be a lack of evidence in support of these contentions. The fact that the mortality-rates from typhoid and phthisis run parallel does not prove that the same cause is operative in both cases. The somewhat excessive number of consumptives who have a history of typhoid does not convince in this direction. It is well known to clinicians that acute tuberculosis very often simulates typhoid in a striking manner, and with all our diagnostic methods it is often very difficult to differentiate the two diseases. In many cases of alleged typhoid preceding phthisis I have been convinced that it was an acute exacerbation of latent tuberculosis which was mistaken for typhoid, just as many attacks of "grippe" are in reality acute exacerbations of chronic or mild forms of phthisis. Typhoid fever, like most other febrile diseases, may, however, activate latent phthisis, which might not have taken an acute or subacute course otherwise. But under the circumstances we cannot consider typhoid *per se* as predisposing to tuberculosis.

OCCUPATION.

Of the factors which have been mentioned as predisposing to, or at times activating, dormant tuberculosis, the character of the occupation of the patients has been given considerable attention by many writers. Considering the important industrial, sociological, and economic bearings of this problem, it is clear that there have been quite some differences of opinion as to the dangers of certain occupations in this regard. John Brownlee, a rational statistician and demographer, says: "With regard to the influence of occupation, a note of warning may perhaps be sounded. The whole field is a very difficult one, and the interpretation of the figures, unless the greatest care is exercised in drawing deductions, not less dangerous than the interpretation of the Holy Scriptures."

The main difficulty is that statisticians commonly take the rough figures from the death reports and calculate the coefficient of mortality according to the occupations of the deceased, without inquiring how long the deceased had been working at the given occupations. Many

[1] Am. Med., N. S., 1914, **11**, 17.

people suffering from chronic phthisis change their occupations, and after death the last occupation is registered, while the one that may really be responsible for the reactivation of the disease is not mentioned.

The process of social selection, which is very pronounced in this respect, is usually not considered. Thus, as has been pointed out by Cobbett, hotel servants in England show a very high tuberculosis mortality. Some would be inclined to attribute it to their indoor life, as well as to their proclivity to drink alcoholic beverages excessively. But others working indoors, in the garment industries, for instance, have not an excessive tuberculosis mortality. No doubt there is a

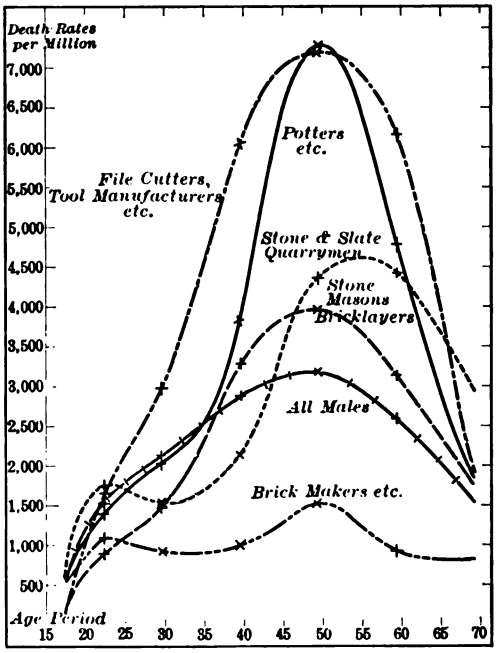


FIG. 19.—Mortality from pulmonary tuberculosis, 1900-1902 in certain occupations in, England and Wales. (Brownlee.)

process of social selection going on. Occupations which do not require excessive muscular exertion are likely to attract the weakly and the sick—among them many with latent or quiescent tuberculosis. For this very reason policemen are less liable to develop tuberculosis—only strong men are taken into the service.

Another illustration is brought by Brownlee.[1] After the age of thirty-five, the mortality from phthisis among persons included in the class of commercial clerks, and in the class of carmen, carriers, etc.,

[1] An Investigation into the Epidemiology of Phthisis in Great Britain and Ireland, London, 1918, p. 15.

are practically identical with the mortality of males in general. Before this age there is considerable excess of phthisis among carmen, etc. It is hardly likely that these variations are to any extent occupational. It is much more probable that the occupations have been originally selected for reasons of physical fitness, or the reverse. This is confirmed when phthisis mortalities among commercial travelers are considered. In this case the mortalities from phthisis are practically identical throughout the general population, though on a slightly lower level. As commercial travelers are largely chosen from the clerk class,

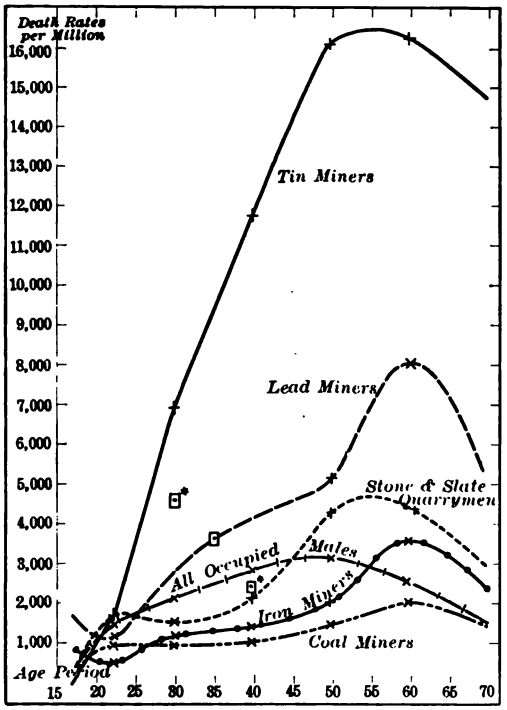


FIG. 20.—Mortality from pulmonary tuberculosis 1900–1902 among miners in England and Wales. (Brownlee.)

it would seem to be indicated that the fit are put upon the road and the unfit kept upon the stool.

Occupations may predispose to tuberculosis by the dust created, and which the workers are compelled to inhale, while working at the industry; by dangerous fumes or odors emanated from the material used; and also, as we shall see later on, by keeping the workers in unclean factories and mills, paying them low wages, etc., all of which reduces their vitality and resisting powers.

Dust as an Etiological Factor in the Evolution of Phthisis.—Because pulmonary tuberculosis has been considered a disease caused mainly

by inhalation of the virus, dust has been mentioned as an etiological factor by writers on the subject for centuries. In nearly all treatises on tuberculosis, or on occupational diseases, it is never omitted to state emphatically that persons pursuing occupations at which they are exposed to the inhalation of mineral, metallic, vegetable, or animal dust are more likely to contract tuberculosis, and die from it, than others. A few figures, culled from Frederick L. Hoffman's recent monograph,[1] will supply drastic illustrations of the great dangers of contracting tuberculosis of those exposed to the inhalation of certain kinds of industrial dust. During 1908 and 1909 the proportionate mortality from pulmonary tuberculosis in the United States Registration Area was 14.9 per cent of all deaths among all occupied males, and only 8.7 per cent among farmers, planters, and farm laborers. Among workers exposed to metallic dust, it was 21 per cent; among engravers, 31.1 per cent; among printers, lithographers, and pressmen, 29.5 per cent; among tool and cutlery makers, 24.1 per cent; among jewelers, 17.8 per cent; among iron and steel workers, 14.9 per cent. Those working in industries exposing them to the inhalation of certain kinds of mineral dust are even more menaced by tuberculosis: 21.3 per cent of the workers at these industries succumbed to this disease; potters, 34.6 per cent; glass-blowers, 32.1 per cent; marble and stone cutters, 30.7 per cent; plasterers, 16.7 per cent; brick and tile makers, 12 per cent; coal miners, 9 per cent. Similar statistical evidence is available for other countries, notably England and Wales, Australia, Germany, France, etc.

Menacing Kinds of Dust.—From the few figures just brought together it is evident that not all kinds of dust are etiologically related to pulmonary tuberculosis. Among coal miners tuberculous phthisis is relatively uncommon, though they undoubtedly inhale large quantities of mineral dust, which almost invariably reaches the deeper respiratory passages, and remains there, as is evident from the frequency of pneumokoniosis among them. Kuban, as far back as 1863, drew attention to this fact and said that in France "coal dust is unable to cause pulmonary tuberculosis, or even to favor the evolution of this disease." In England, Oliver shows that the same condition prevails, and Brownlee says that "phthisis is a comparatively uncommon disease among coal miners." Newsholme brings evidence to the effect that coal miners in Derby and Nottinghamshire stand high on the list as to expectation of life, being only surpassed by farmers, agricultural laborers, teachers, etc. In the United States available evidence is to the same effect. Carr, whose experience among miners in the largest coal mining towns in Southern Illinois extended over twenty-five years said that "bituminous coal miners, while actively engaged in their occupation, are practically immune from tuberculous disease." He inquired among 600 practising physicians in the coal mining towns of

[1] Mortality from Respiratory Diseases in Dusty Trades, Washington, 1918.

this country and, out of some 200 replies received, about 75 per cent sustained the point of view that "during active service in his occupation the bituminous coal miner is immune to tuberculosis, wholly, or in part." The reservation "during active service," of course, indicates that the immunity is limited, because tuberculous persons leave the mines. But available evidence seems to show that tuberculosis is not excessively common among coal miners, despite the fact that coal dust penetrates the deep respiratory passages.

More noteworthy is it that street-sweepers and coachmen, in spite of exposure to excessive inhalation of dust, are not excessively liable to phthisis. Cornet concluded from this fact that the dangers of tuberculous infection in the street are *nil*. Sommerfeld has shown that in Berlin the street-sweepers have only half the rate of mortality from phthisis when compared with the mortality of the working classes in that city. In New York City, where several years ago considerable agitation was made in favor of protecting the street-sweepers against an excessive morbidity and mortality from tuberculosis, statistics have not borne out these contentions. Hoffman's statistics, gathered for a monograph on the excessive mortality from consumption in occupations exposed to municipal and general dust, show clearly that "the recorded mortality from consumption among men in this employment is not decidedly excessive."

Another kind of dust which is apparently harmless in regard to phthisiogenesis is limestone, and also plaster of Paris. In England it has been found, according to Collis,[1] that masons in districts where limestone is worked do not suffer from phthisis in excess, while masons in districts where sandstone is worked are peculiarly liable to succumb to this disease, and have a shorter prospect of life. Halter and Garb have recorded the same to be a fact in Germany, and Fisac[2] reports that in Spain the workers in quicklime and plaster of Paris are immune to tuberculosis despite the fact that they live in squalid dwellings, and are underfed. He believes that their immunity is due to the inhalation of dust containing lime. Hoffman[3] brings considerable evidence to this effect from among the workers in lime in the United States. Several writers have suggested the utilization of lime in the therapeutics of tuberculosis.

Effects of Inhaled Dust on the Pulmonary Tissue.—Barring the few kinds of dust just mentioned, it appears that the inhalation of dust is a strong predisposing factor in the evolution of tuberculous lung disease. In considering the menace of dust it must first be taken into consideration that nature has placed many barriers in the way of even fine dust entering the deep respiratory passage with the inspired air (see p. 50); even when reaching the mucous membranes of the bronchi and lungs the latter are not very tolerant of foreign bodies, and most

[1] Public Health, 1915, **28**, 252, 292.
[2] Rev. de hyg. de tuberc., 1909, **5**, No. 54.
[3] Loc. cit., p. 217.

of it is soon expelled with the cough and expectoration which it provokes. However, Moritz found that the sensibility of the mucous membrane of the respiratory tract, from the nose to the trachea, is reduced in persons working as grinders in a steel factory in Germany. Large masses of metallic dust could be seen lying on the vocal cords and the mucous membrane of the trachea without provoking cough to expel it. For this reason some dust remains, and it is taken by the lymph channels and carried farther on. But after persistent deposits of dust in the alveoli, the irritation it produces excites a reactive inflammation, clogs the lymph channels, and lowers the resisting powers of the invaded lung, preparing the soil for the deposit of tubercle bacilli which may thrive in defective tissues. The glands of the lungs normally act as filters which retain the dust brought in by inhalation, but if new deposits are repeatedly brought into these glands, they are ultimately damaging to the structure and function of the lung, impairing the glands as filters.

On the other hand, Lubarsch[1] is of the opinion that clogging of the pulmonary lymphatics by coal dust is preventive of tuberculosis for another reason. The devitalized tissue is a poor soil for the growth of the microörganisms. Then, bearing in mind that extension of tuberculous lesions in the lungs commonly takes place along the lymph channels, it is clear that when these are damaged and clogged, they prevent the extension of the pathological process. He points out that the process is thus purely mechanical, and not biochemical. In a certain coal region where he found that the miners show only one-half the tuberculosis mortality-rate when compared with the general population, this is only true of pulmonary tuberculosis. Tuberculosis of the bones and joints is just as frequent among them as among others.

This may be true of pneumokoniosis produced by coal dust. But in the case of other forms of dust, other factors are at work. In some cases the mechanical factor is again responsible, while in others we have recently been convinced that biochemical reactions take place. Collis,[2] in his "Milroy Lectures of 1915," as well as the experimental investigations of Mavrogordato,[3] the statistical investigations of Brownlee, Hoffman, and others, have shown that the most dangerous dust is that containing crystalline silica. The same is true to a certain degree of metallic dust, excepting perhaps iron dust. As to why coal dust, lime, plaster of Paris, etc., should be harmless in this regard, while flint, slate, tin, copper, etc., do promote the development of pulmonary tuberculosis, has not been explained satisfactorily. Experimental investigations of Mavrogordato tends to show that it mainly depends on the promptness with which various kinds of dust are eliminated from the lungs. It appears that dust entering the lungs excites an inflammation resulting in shedding of epithelial cells lining the air

[1] Ztschr. f. Aerztl. Fortbildung, 1918, **15**, 39.
[2] Public Health, 1915, **28**, 252, 292; **29**, 54.
[3] Jour. of Hygiene, 1918, **17**, 439.

passages. These cells pick up the dust and remove it promptly in cases of certain kinds of dust, as coal dust; while in the case of other kinds, as flint, or quartzite, this process of elimination is slow, or wanting, and the foreign bodies remain within the lungs for months. Experimenting with lamp black and pulverized glass, Corper[1] found that glass inhaled by guinea-pigs has a markedly accelerating influence, while lamp black has a retarding effect.

Recent investigations have revealed another important point. It appears that the process is not entirely mechanical, but that biochemical phenomena play an even more important rôle. It has been shown that dust inhalation is dangerous in proportion to the amount of quartz, or silica, it contains. But Haldane found that despite the fact that shale contains as much as 35 per cent of silicates (but not crystalline silica) it does not induce phthisis when inhaled for years by miners in England. Likewise, Brownlee found that the phthisis mortalities among lead, iron, and coal miners reaches its maximum between the ages of fifty-five and sixty-five years, or ten years later than that found among silica workers, or males in general. With coal miners, among whom phthisis is a comparatively uncommon disease, it is at this age that the only approach to the level of the mortality among males in general is made. Among ironstone miners there is about twice the mortality of the coal miners at this age, and among lead miners about four times. He finds that while there is very little phthisis during early age among the general population, there is also very little among the miners, whereas, wherever early phthisis is common, it is also common among miners. On the other hand, Collis[2] points out that a form of tuberculosis with an age incidence conforming to Brownlee's middle-age type is extraordinarily prevalent among those who inhale dust of silica but not any other material. Those masons and quarrymen who work freestones suffer, but not those who work limestones; makers of silica suffer, but not makers of ordinary bricks; men who grind metal articles on sandstone wheels suffer, but not those who grind on carborundum or emery wheels; men who crush flint into fine flour suffer, but not those who crush emery or glass; miners who work mineral veins of tin, gold, or lead found in quartzite "country rock" suffer, but not those where country rock is of slate, or other non-silica material.

In order to produce its deadly effect, silica must be present as uncombined SiO_2; if it exists as a silicate, *e. g.*, in clays, the dust is not thus harmful. Collis suggests the following explanation why silica dust predisposes to lung disease: The chemical similarity between silica and carbon, as shown by their proximity in the periodic table, and their tendency to form colloid compounds may be the determining factor, permitting silica to replace carbon in the colloid complex of living protoplasm with the formation of new tissue, the tissue of the silicotic

[1] Am. Rev. Tubercul., 1919, **3**, 605.
[2] Tubercle, 1920, **1**, 54.

nodule which, as Watkins-Pitchford has pointed out, is histologically so suggestive of fibrosarcomatous growth. Collis thus suspected that the process known as "silicosis" is effective in producing a definite chemical reaction in lung tissue which renders them a ready prey to the tubercle bacilli.

More recent investigations by W. E. Gye[1] have confirmed the suspicions that biochemical reactions are responsible for the development of tubercle. To begin with, Gye and Cramer found that the tetanus bacilli are pathogenic only when introduced into the body together with silica. If the soil in which the bacilli are carried does not contain soluble calcium salts of hydrated soluble silica, tetanus bacilli are harmless. They found that mice are sensitized to tetanus with silicic acid. Pursuing along the lines, Gye and Kettle have shown that silicic acid, and even silica dust, also sensitize these animals to tuberculosis, which spreads in the tissues injured by silica. That particles of silica disintegrate in the lungs into silicic acid has been shown by W. Watkins-Pitchford.

These biochemical reactions are worthy of further study. They seem to open up a new field for investigations as to why, in most cases, tuberculous infection is more or less harmless, while in others it produces deadly effects. But the field has hardly been scratched.

Inhalation of Noxious Fumes.—Occupations involving the inhalation of noxious fumes have at various times been considered etiologically related to tuberculous disease. Statistical evidence has not been in the direction of supporting this contention. But during the recent World War, when thousands of soldiers were attacked with various poisonous gases, it was found that while many of the survivors suffered from various pulmonary diseases, very few developed tuberculosis. Here we had an extensive experiment testing the etiological relationship of noxious gases to phthisis in human beings. Evidently it proved etiologically negative. Numerous cases seen by the writer, though showing symptoms and signs suggestive of tuberculous disease, have proved on careful and prolonged observation to be free from it.

Social and Economic Conditions.—It thus appears from the brief survey of the subject that occupation *per se* cannot be considered as specifically predisposing to tuberculous disease, with the exception of those which involve exposure to, and inhalation of, metallic and certain kinds of mineral dust. But even in these occupations there are significant exceptions, as we saw in the case with street, iron, and coal dust, limestone, plaster of Paris, etc.

There has, however, been found a correlation between the economic conditions of the workers and the incidence of tuberculosis among them. Economic conditions may be gauged by the wages paid to workmen. B. S. Warren,[2] in a study of conditions among the workers in the United States Government printing and engraving plants, found

[1] See Dale, Lancet, 1921, **2**, 112.
[2] Tr. Nat. Tuberc. Assn., 1913, **9**, 153.

that despite the fact that they are badly overcrowded, with poor ventilation, etc., the mortality from tuberculosis is rather low among the employees. He assigns as a reason the good wages paid by the Government to these workers. He finds from census statistics that low wages go hand-in-hand with high tuberculosis mortalities. The difference in wages or income means a difference in nutrition, social contentment, and general welfare, which renders the farm laborer more susceptible to phthisis than his employer, the cotton-mill operative more than the general population. Likewise, he finds that of deaths among males reported by the Census Bureau for 1909, giving the occupation of the deceased, 14.7 per cent were from tuberculosis, as against 20.9 per cent among females. The reasons for this disparity are many, but undoubtedly the inadequate wages paid to women are responsible for a considerable portion of phthisis among female workers. The enormous increase of tuberculosis in European countries during and soon after the World War (see p. 89) points in the sam direction. Another fact in confirmation is the sharp increase in the morbidity and mortality from tuberculosis during the war in Europe, and its quick recession as soon as food has become more abundant.

CHAPTER V.

PHTHISIOGENESIS. II.

THE PHENOMENA OF IMMUNITY.

Experimental Tuberculosis vs. Spontaneous Phthisis.—Infecting experimentally an animal with tubercle bacilli, we know exactly what morbid phenomena to expect. On injecting into the peritoneal cavity of a guinea-pig a certain quantity of the pure culture of tubercle bacilli, tuberculous peritonitis soon develops, followed by tuberculosis of other organs—the spleen, the liver, the lungs, the kidneys, etc., until it finally succumbs. But what will happen after a human being is infected in the usual spontaneous manner we cannot prognosticate with any degree of certainty. The individual may pass through life without showing any morbid manifestations which can be attributed to the infection. In fact, the vast majority of people have been infected during their childhood and are none the worse for their experience, as has already been shown. A large proportion of those in whom distinct lesions of a tuberculous character have been found at the autopsy knew nothing about it during their life. On the other hand, in a certain, and it must be said a relatively small proportion, the infection is followed, sooner or later, by symptoms of some clinical form of tuberculosis.

This is, however, not the only difference between experimental tuberculosis and spontaneous phthisis as we meet it in human beings.

It appears that *phthisis is a disease met with exclusively in human beings and rarely, if ever, in the lower animals; certainly not in animals which have been infected experimentally in the laboratory, be it by inoculation, ingestion or inhalation of tubercle bacilli.* In guinea-pigs, rabbits, etc., in whom spontaneous tuberculosis is exceedingly rare, only nodular tubercles, consisting of avascular, cellular masses, are formed after experimental infection; while spontaneous human phthisis is mainly a productive and exudative inflammatory process of the lungs in which there may, or may not, be any of the characteristic tuberculous cell-proliferation. In other words, experimental infection in animals results in general or miliary tuberculosis, a disease but rarely met with in humans. "Real pulmonary tuberculosis," says von Hansemann,[1] a pathologist of vast experience, "in the anatomical sense, is always part and parcel of general tuberculosis of all the organs in the body. Pure and isolated pulmonary tuberculosis in the anatomical

[1] Berl. klin. Wchnschr., 1911, **48**, 1.

sense, *i. e.*, in which there are no other tuberculous changes in the lungs than the development of submiliary tubercles, never occurs so far as my experience goes. But it is a noteworthy fact that from this disease, which in reality alone deserves the name pulmonary tuberculosis, phthisis never evolves. I know of no case in my own experience, nor from medical literature, in which the disease began as acute miliary tuberculosis in the anatomical sense, and then turned into pulmonary phthisis." But phthisis may be, and quite often is, complicated by general miliary tuberculosis. This frequently occurs before the fatal termination of the case.

In the same sense we find that Ribbert[1] makes a sharp distinction between experimental tuberculosis in animals, and phthisis in human beings: "It is undoubtedly a fact that tubercles may be produced in the lungs of animals which are made to inhale dust containing tubercle bacilli. But, (1) the disease thus produced is not the same as that in human beings; (2) we cannot, without further proof, conclude that human beings are infected in the same manner. The conditions under which humans inhale tubercle bacilli are, at least from the viewpoint of quantity, distinctly different from those prevailing during experimentation. It can neither be proved that individuals always inhaled tubercle bacilli before becoming sick, nor that the latter settled primarily in the particular organ in which they proliferated. Neither the clinical nor the anatomical findings sufficiently support this view. It is self-understood that I do not in the least deny that in man also disease may directly follow the inhalation of tubercle bacilli, but it is a question how often this takes place. From mere possibility to uncontrovertible proof which will cover all tuberculous diseases of the lungs, is quite a distance." "Pulmonary phthisis," says Bacmeister,[2] "is a disease found exclusively in adult human beings; it never occurs spontaneously in animals, nor has it ever been produced experimentally."

Before applying unequivocally experimental findings to man we must first demand that infection of animals should result in isolated apical lesions which extend gradually downward in the lung in the typical chronic manner. All other forms of tuberculosis which are produced experimentally in the lungs of animals do not prove much, because their morbid anatomy diverges considerably from the changes found in human phthisis.

The problem why the human adult, after infection with tubercle bacilli, develops phthisis, a disease unknown in early childhood and among the lower animals, has not yet been solved to the satisfaction of all who are entitled to an opinion. Freund, Hart, Bacmeister, and others believe that pressure of a short rib or an ossified first costal cartilage upon the apex of the lung is responsible for the apical localization of phthisis (see p. 118). We have, however, shown that this theory does not explain everything connected with the problem.

[1] Die Ausbreitung der Tuberkulose im Körper, Marburg, 1900.
[2] Die Entstehung der menschlichen Lungenphthise, Berlin, 1914, p. 35.

Various other theories have been promulgated to explain the origin of human phthisis.

Intensity of the Infection.—After tubercle bacilli have succeeded in gaining an entrance into the body, they cause disease only when the initial dose is sufficiently large and virulent, and they gain a foothold on susceptible tissues. Experience with most microbic diseases has shown that the average animal can withstand the entry of certain minimal doses without becoming sick with the specific disease. It is clear that in spontaneous infections we are not in a position to know the infecting dose. Experimentally it has been found that when the dose is small the animal may escape disease, though some authors report that very few germs, even one bacillus according to Römer, C. Fraenkel and E. Baumann,[1] may cause disease. Cobbett found that small doses of bovine bacilli, when injected into calves, produce only localized and limited lesions which soon become fibrous and calcareous, and thus assumed a retrogressive type; while the animals themselves, after transient disturbance of health, remained in excellent condition up to the time when they came to be slaughtered and examined. Medium doses (10 mg.), on the other hand, produced irregular results, while larger ones (50 mg.) invariably caused generalized tuberculosis which, in all but few animals (6 per cent), proved fatal within a few weeks or months (seventeen to seventy-six days). Gilbert and Gregg found it required between 10 and 120 bacilli to infect a guinea-pig. Webb and Gilbert found that this number of bacilli was sufficient to cause infection, but not disease, in a human child. H. J. Corper showed that subcutaneous injection of 0.000001 mg. of moist culture produces tuberculosis in a guinea-pig within two months, while smaller doses usually produced only local lesions. Large doses produced multiple foci in various parts of the body. Employing a new method, Hugo Selter[2] recently found that one bacillus is enough to infect a guinea-pig by the subcutaneous and intravenous routes, and even by inhalation. When a larger dose is employed, it makes little difference as to the resulting disease, duration of life, etc., of the animal whether 100, or 1000, 10,000 or 100,000 bacilli are used, provided that virus is injected intravenously, subcutaneously, or intraperitoneally. Thoeni and Thaysen,[3] however, could not confirm these findings. They found that 10 to 343 bacilli are insufficient to infect an animal when administered subcutaneously or intraperitoneally. They attribute the success of others to defective methods in counting the bacilli.

Many writers have pointed out that there are significant differences in the result obtained in experimental infections according to the route by which the bacilli are brought into the body. Findel,[4] while exposing 87 guinea-pigs to inhalation of tubercle bacilli, found that when a very

[1] Ztschr. f. Hygiene, 1906, p. 247.
[2] Veröffentl. Rob. Koch Stiftung, 1916, parts 11–12, p. 105.
[3] Zblt. f. Bakteriol., 1916, **77**, 308.
[4] Ztschr. f. Hygiene, 1907, **57**, 104.

small number of the microörganisms, less than 50, were inhaled, disease results, and in young animals which are more susceptible than older ones, a single bacillus may produce a tuberculous lesion. On the other hand, enormous numbers of bacilli are necessary to produce infection by ingestion: Findel found that when 19,000 to 382,000 bacilli were given to 14 guinea-pigs with food, no tuberculous lesions could be discovered after observing them for as long as one hundred and seventy-four days. His experience has taught him that infection by ingestion can only be accomplished when the animal consumes at least 10 mg. of a pure culture of tubercle bacilli (about 35,000,000 bacilli). It thus appears that infection by ingestion is accomplished only when the infecting dose is about six million times greater than that required for infection by inhalation.

These interesting experimental findings cannot be applied without reservations to spontaneous human infection. These experiments were made with pure cultures of bacilli, and human beings hardly ever experience the entry of pure cultures by any route. Inhaled, ingested, or inoculated, the virus is always carried in dust, sputum, food, etc., together with various additional bacteria which may have an immense influence on the outcome. Moreover, we have seen that human beings display great resistance to tuberculous infection, even during infancy and childhood, and not all infections are followed by disease, and there are reasons to believe that only repeated infections with small doses of the virus, or a single very large one, is necessary to produce progressive tuberculous disease.[1]

It would be rash to conclude that such large doses as would be necessary to infect experimentally an animal of the size of a human being are ever inhaled even in the proximity of a coughing consumptive; it appears that in most cases of such infections the dose is too small to produce disease. On the other hand, it is known that the bacilli multiply in the human body, and the few introduced, finding suitable conditions for life, may proliferate and produce disease of any magnitude. Cobbett is inclined to attribute the harmlessness of small doses of bacilli to the following factors: After a minimal dose of bacilli enters the body, the organism at once begins to mobilize and develop its protective forces which are sufficient to deal with a few bacilli, while when a larger number is introduced, it may overwhelm the natural protective forces. This is confirmed to a certain degree by clinical observations in children. Most are infected with small doses of tubercle bacilli in early life, and are hardly harmed by the infection. A small proportion, particularly those subjected to massive infection, succumb to acute tuberculous disease of some form. The mild infections enhance the work of the protective apparatus and prevent the multiplication of the bacteria; large doses can cope with the slight amounts of immu-

[1] For a complete review of the literature on this subject, see E. R. Baldwin and L. N. Gardner, Am. Review of Tubercul., 1921, 5, 429.

nizing bodies which they provoke, and can keep on multiplying and destroy vital tissues.

This may explain the immunity of children living in modern communities in which tubercle bacilli are ubiquitous. But it does not explain the development of phthisis in the adult who has been infected in early life with minimal doses of tubercle bacilli which remained latent for many years. The latter has been explained again by the various theories of *predisposition*, or *diathesis*, the innate, or inborn, tendency of some persons to acquire diseases which depends on certain peculiarities of the structure and function of the various organs of the body, all of which has been discussed in the preceding chapter.

Phthisis Acquired during Childhood.—During recent years the theory that phthisis is a late manifestation of tuberculosis acquired during childhood has been gaining ground. Behring,[1] basing his opinions on experiments with guinea-pigs, maintains that a single infection cannot result in phthisis. He says that phthisis is the result of reinfection of a person who was already once infected during infancy, mainly through deglutition of milk derived from tuberculous cows. The bacilli pass through the gastro-intestinal tract into the lymphatics where they remain for years in an avirulent or mildly virulent state, and in the adult, as a result of some intercurrent affection, they become again virulent and cause phthisis. "Phthisis is but the last verse of the song, the first verse of which was sung to the infant at its cradle."[2]

Hamburger's[3] conception of phthisis is also that it must not necessarily be preceded by recent infection, but that it is rather a reawakening, or an exacerbation, of an old, "healed," or latent tuberculous process. He points out that tuberculosis runs a different course in children from that in adults—pulmonary phthisis which is so frequent in adults is exceedingly rare in children. But we know that most people have passed through a tuberculous infection during childhood. Under the circumstances the inference is justified that pulmonary phthisis is invariably preceded by a tuberculous infection many years before its onset.

To Hamburger the course of phthisis is similar to that of syphilis, with periods of health and quiescence, or latency, interrupted, or followed by periods of acute or subacute exacerbations. The primary lesion is inoculated during childhood, before the individual reaches his tenth year of life. During infancy this primary focus, if massive infection has taken place, or the resistance is low, may cause miliary tuberculosis, or hematogenous metastasis, but in the vast majority of people it heals, or remains dormant. In those in whom metastatic deposits of tubercle bacilli are distributed in various parts of the body, secondary tuberculous manifestations make their appearance, consisting in tuber-

[1] Deut. med. Wchnschr., 1903, **29**, 689; British Med. Jour., 1903, **2**, 993.

[2] Einführing in die Lehre von der Bekämpfung der Infektionskrankheiten, Berlin, 1912, p. 354.

[3] Die Tuberkulose des Kindesalters, Leipzig, 1912.

culosis of the glands, bones, joints, meninges, etc. After the tenth year the tertiary manifestations are met with, consisting in the various forms of chronic pulmonary phthisis, tuberculosis of the larynx, tumor albus, certain cases of joint diseases, of the kidneys, lupus vulgaris, tuberculous iritis, adhesive pleurisy, etc. These last are practically never seen in infancy and early childhood; only after the disease has lasted for many years they may appear, just as the late manifestations of syphilis—tabes, general paralysis, etc., are only rarely seen in early youth, although syphilis is quite frequent at that period of life.

Phthisis is thus, according to Hamburger, an exacerbation of tuberculosis which has been acquired during early childhood and remained latent for many years until some exciting cause, or a reduction in the powers of resistance, has brought about conditions favorable for its development.

Latency of Tuberculous Lesions.—Before concluding that in the adult phthisis is an exacerbation of an infection with tubercle bacilli which has taken place during childhood, it must be ascertained whether living and virulent tubercle bacilli can remain within the body for many years without producing symptoms of disease. We have seen (Chapter III) that autopsy findings of persons who died from any cause, and tuberculin tests in the living have proved conclusively that 60 to 95 per cent of human beings have tuberculous lesions in some parts of their bodies, and the majority remain healthy. We have also shown that the number of such latent lesions is very small during the first year of life, but increases annually, so that when reaching adolescence, the percentage harboring tuberculous lesions reaches over ninety.

Animal experimentation has brought forward another important fact: Tubercle bacilli may remain for a long time in the tissues without producing gross, or even microscopical changes. Bartel, Weichselbaum, Macfadyen and MacConkey,[1] Goodale,[2] Rosenberger,[3] Harbitz,[4] and others, have found this to be true in human beings. Bartel[5] refers to this condition as the *lymphoid latency* of tuberculosis. He kept guinea-pigs in dwellings inhabited by consumptives, allowing them to roam around freely in the rooms and playing with the patients for several weeks. They were then kept under observation until their natural death, or killed when symptoms of disease were noted in them. At the autopsies it was found that of 27 animals, 17, though showing no gross pathological changes, had mesenteric and bronchial glands which were pathogenic when inoculated into animals. Similar observations have been made when human glands, removed surgically, or postmortem, were examined: No gross or even microscopic changes could be found, but when properly inoculated into animals, tuberculosis was produced;

[1] British Med. Jour., 1903, **2**, 129.
[2] Boston Med. and Surg. Jour., 1906, **155**, 632.
[3] Am. Jour. Med. Sci., 1905, **130**, 95.
[4] Jour. Infect. Dis., 1905, **2**, 143.
[5] Wien. klin. Wchnschr., 1905, **18**, 218; 1906, **19**, 25; 1907, **20**, 1149.

in some instances tubercle bacilli were even cultivated from such apparently healthy glands. As far back as 1890, Loomis found that in non-tuberculous persons 26.6 per cent were latent carriers of tubercle bacilli; Calmette[1] reports even higher percentages of tubercle bacilli in a latent stage, without any traces of pulmonary lesions in infants who died in hospitals for non-tuberculous patients, and who presented no symptoms of tuberculous disease, and he concludes that glandular infections have usually a tendency to remain latent.

This lymphoid latency of tuberculosis may last for many years; in fact, for the natural life of the individual. But with advancing age, localization takes place and gross, as well as microscopic, changes of the affected structures takes place. Numerous lesions found in the glands, lungs, and pleuræ of persons who died from other causes contain living and virulent tubercle bacilli, though during life no symptoms of this disease have been observed. This is observed not only in caseated lesions, but also in the fibroid, and even the calcified nodules found in adults. In other words, calcification of a tuberculous lesion does not kill the bacteria responsible for the pathological process. Investigations by Piéttre, Uhlenbrock, Rabinowitsch,[2] Lubarsch, Calmette, Guérin and Deléarde, and many others, showed that the chalky deposits found in the bronchial and mesenteric glands, in human beings as well as in animals, contain virulent tubercle bacilli. Calcified nodules are found mainly in adults, most commonly in persons over forty; they are hardly ever seen in infants under two years of age. While some of these nodules are completely surrounded by fibrous capsules, the majority are not, and at any time, when resistance is at a low ebb, the bacilli may invade the blood or lymph stream, and thus be carried to some vital organ where they proliferate and cause disease. While they may thus cause metastatic auto-infection, they ordinarily serve a good purpose in immunization, as we shall soon see.

Considering the wide prevalence of this sort of tuberculous lesion, and that tubercle bacilli may remain in the tissues for years without causing any obvious anatomical changes, there is justification for the assumption of latent tuberculosis. For the clinician it is important to bear in mind that these latent lesions require no treatment, as a rule. When the equilibrium between the immunity conferred by these latent bacilli, on the one hand, and natural resistance of the carrier, on the other, is disturbed for any known or unknown reason, tuberculous disease results from these bacilli. The form of the disease that may result from this metastatic auto-infection, or the organ that may be attacked, seems to depend more on the constitutional peculiarities of the patient than on the source of the virus. There are analogous conditions in pathology. The tertiary manifestations of syphilis are also caused by metastatic auto-infection from latent lesions; in malaria similar phenomena are observed. In leprosy a latency of twenty years

[1] L'infection bacilliare, Paris, 1920, p. 110.
[2] Berl. klin. Wchnschr., 1907, **44**, 35; Ztschr. f. Tuberkul., 1910, **15**, 217.

and more after the infection is common. In fact, in this disease, there have been observed cases in which infection took place many years before the outbreak of the symptoms, and no chances for reinfection had been available during the long period of latency.

Immunity or Allergy.—The view of phthisiogenesis which has been gaining ground of late, and which apparently is based on a sound foundation, has been formulated by Paul Römer, to the effect that *phthisis is a manifestation of immunity against tuberculosis which has been acquired by an infection during early childhood.*

It appears that the observations made in most of the transmissible diseases that one attack renders the individual immune against renewed infection with the same virus, hold good in tuberculosis; *mild infections during childhood endow the organism with a certain amount of immunity against further and renewed exogenous infection with tubercle bacilli.* An individual with a healed or latent lesion, acquired during early childhood, is immune to these microörganisms. Repeated infections with the same virus may be reinfection or superinfection. By superinfection is understood a second infection at a time when the lesions produced by the first infection have not healed, while reinfection implies a new infection when the lesions produced by the first have completely healed. "Inasmuch as we may accept as a great probability that in tuberculosis healing in the strict scientific sense never occurs," says Hamburger,[1] "all repeated infections in tuberculosis are to be considered superinfections." We use the word *reinfection* because this term has gained extensive currency in medical literature.

Experimental Proofs of Immunity.—Experimentally acquired immunity by an inoculation of tuberculosis has been proved to exist by the researches of Koch,[2] and then confirmed by Behring, Römer,[3] Hamburger, Webb and Williams,[4] Rossignol, Krause and Volk, Allen K. Krause,[5] and many others. When a healthy guinea-pig is inoculated with tubercle bacilli in pure culture, the wound closes within a couple of days and seemingly heals. But about ten to fourteen days later there appears at the site of the inoculation a hard nodule which soon breaks down, leaving an ulcer which presists until the animal dies. It is different when a tuberculous animal is inoculated with tubercle bacilli. The wound also heals, but no nodule is formed and a few days later the point of inoculation becomes indurated, dark in color all around the punctured point to about 1 cm. in diameter. During the next few days the spot becomes necrotic and the involved tissues are shed, leaving a flat ulcerated area which usually heals quickly, and permanently. Moreover, while after infecting a healthy animal the regional lymph glands become swollen, this does not occur after reinfection of a tuberculous animal.

[1] Med. Klinik, 1915, **11**, 34. [2] Deutsch. med. Wchnschr., 1891, **17**, 101.
[3] Beitr. z. Klin. d. Tuberk., 1910, **17**, 287; 1912, **22**, 301.
[4] Jour. Med. Research, 1911, **24**, 1.
[5] Am. Rev. Tubercul., 1919, **3**, 1.

The work of Römer[1] and Hamburger[2] along these lines has modified our conception of tuberculous infection, and suggested prophylactic measures which are actually revolutionary. They have found that reinfection is as difficult and even as impossible in tuberculosis as in syphilis. All modes of infection were tried, inoculation, feeding, inhalation of tubercle bacilli in dust or spray, and contact infection, which are akin to the usual modes of spontaneous infection in human beings, but no new tuberculous lesion could be produced in tuberculous animals, while healthy controls were infected and succumbed to the disease in some form. Not only were guinea-pigs and rabbits—which are very susceptible—thus tried, but sheep which are not as vulnerable to tubercle bacilli, and also dogs which are strongly refractory, and monkeys which display the same degree of susceptibility as man. Römer found that when a healthy sheep is infected with a certain dose of tubercle bacilli, it succumbs within eight weeks to acute pulmonary tuberculosis, but the same dose is harmless in a tuberculous sheep. In monkeys the results were the same. Hamburger and Toyofuko have proved that infected guinea-pigs are not only immune to inoculation, but also to inhalation, which is deadly to healthy control animals. It appears from Römer's studies that this immunity is not transmitted by heredity, even when displayed by pregnant mothers.

It has also been found that this immunity is not only true of exogenous superinfection, or additional infection with bacilli of other strains, but also of superinfection with bacilli taken from their own lesions.

Another important point was established by the experimental investigations of Römer and Hamburger: If the reinfecting dose of tubercle bacilli is small, perfect immunity is found—the point of inoculation heals quite soon. As a rule, the immunity is observed in animals which have been tuberculous for some time, three or four months. But if the reinfecting dose of tubercle bacilli is massive, it soon causes death of the animal.

The results of these experimental researches are well founded, having been confirmed by many workers in various countries, so that at present they are as firmly established as anything else we know about spontaneous and experimental tuberculous infection. But there arise several problems of immense interest in our study of phthisiogenesis. Knowing well that the vast majority of human beings have been infected with tubercle bacilli during childhood, even those who have no clinical evidence of phthisis, we may justly ask, Can adults be infected with tuberculosis at all? The bearings of this problem on prophylaxis are enormous. How does phthisis develop from lesions acquired during infancy and childhood? Is it due to a second infection immediately before the onset of the disease, or do the old, hitherto dormant lesions for some reason flare up, begin to extend and produce metastasis?

[1] Beitr. z. Klin. d. Tuberk., 1910, **17**, 287, 383; 1912, **22**, 265, 301.
[2] Ibid., 1910, **16**, 271.

Modes of Reinfection in Human Beings.—A person who has once been infected with tubercle bacilli may be reinfected with the germs which he harbors within the body, or with bacilli which have grown in the body of some other person, or in an animal. In the case of endogenous or autogenous reinfection the process may be very simple: A softened tuberculous lesion in the lung breaks into a bronchus, and during cough the tuberculous material is carried along the bronchial tree to some other part of the lung where it is deposited and, taking root, it produces a new lesion. In this manner there may also be produced laryngeal and intestinal tuberculosis, the latter from swallowed sputum. But endogenous reinfection is not always bronchogenous; it may also be hematogenous—a tuberculous lesion may break into a bloodvessel and then bacilli are carried to various parts of the body; or it may be lymphogenous; the tuberculous material is carried by the lymphatics, infecting the lymph glands, etc.

Exogenous reinfection should be very common, if it takes place at all. The bacilli are ubiquitous, and one suffering from any form of tuberculosis is evidently predisposed, otherwise he would have escaped the disease, despite the first infection. Infection is exceedingly easy, as is evident from the fact that when a child free from tuberculosis is brought in contact with a consumptive, it is soon infected. Hamburger even reports a case where exposure of an infant for one hour was effective in infecting it. We also see this to be a fact in adults: When individuals free from tuberculous infection dating back to childhood, as is the case with primitive peoples, come into contact with tuberculous people, they are soon infected, and succumb in a short time.

Granting these premises, which are based on carefully observed facts, we may be able to study the problem of reinfection in man clinically, even though the experimental method is, for obvious reasons, closed to us. All we have to do is to inquire into the frequency of exogenous and endogenous superinfection and reinfection in tuberculous patients who are inmates in hospitals for consumptives; the frequency of tuberculosis among those who are apparently healthy but live with consumptives; and also the effects of tuberculous infection on persons who are known to have escaped infection during childhood.

Reinfection in Hospitals for Consumptives.—Clinical experience has shown that it is extremely rare that a person should have one of the exanthemata twice during his life. It has also been observed that in a ward filled with cases of scarlet fever, smallpox, etc., there is no danger that patients suffering from the more malignant types of the disease should transmit the virus to those who are passing through a mild or abortive attack of the same disease. In nearly all contagious and infectious diseases we find that during the existence of the malady the patient is immune against exogenous reinfection with the virus of the same disease. The same is true of the exceedingly chronic transmissible disease, syphilis.

Experience in hospitals harboring large numbers of consumptives

should give us information along these lines about tuberculosis. Here the patients have all the opportunities for superinfection with bacilli derived from other patients. For it must be agreed that despite the scrupulous cleanliness observed at present in sanatoriums and hospitals, it is impossible to avoid droplet infection when many patients are brought into intimate contact. In fact, caged guinea-pigs kept in scrupulously clean wards soon contract tuberculosis.

It has, however, never been observed that a mildly infected patient or one in whom the disease has been "arrested," living in an institution should be reinfected from one severely infected and in whom the disease is active and progressive, who shares the ward with him, even when the latter expectorates myriads of virulent bacilli, and offers exceptional opportunities for droplet infection.

Many non-tuberculous patients remain in sanatoriums for months, yet it has not been observed that one should become tuberculous because of his sojourn in the hospital. This is the reason why hospitals and sanatoriums do not separate the "open" from the "closed" cases, *i. e.*, those who expectorate sputum containing tubercle bacilli from those who do not, in spite of the fact that many physicians are convinced that droplet infection is a potent factor in disseminating tuberculosis.

The hospital staff, including physicians, especially laryngologists, nurses, orderlies, etc., come in close contact with the patients in sanatoriums and should become infected if adults, presumably infected during childhood, could be reinfected with tubercle bacilli. But, if the experience of thousands of people in these callings counts for anything, they do not show a higher mortality nor morbidity from tuberculosis than persons in other occupations. The first statistics bearing on this problem were published by C. Theodore Williams,[1] who showed that long before the discovery of the tubercle bacillus, and before any precautions were taken to prevent the transmission of the disease, no case of infection of the hospital staff had been observed. From 1846, when the Brompton Hospital for Consumptives was opened in London, to 1882 "statistics showing that among the physicians, assistant physicians, hospital clerks, nurses and others, to the number of several hundred, who had served in the hopsital (not few of them having lived in it for a number of years continuously), phthisis had not been more common than it may be expected to be on the average among the civil population of the town." In a later paper Williams[2] brought these statistics down to 1909 and found that conditions remained the same. But while during recent years the rarity of phthisis in the hospital staff, may be attributed to the improvement in the hygienic conditions, and the disinfection of sputum, this cannot be said to have been operative before 1882. During the thirty years of the existence of the Montefiore Hospital, no nurse, orderly, or physician has been observed

[1] British Med. Jour., 1882, p. 618.
[2] Ibid., 1909, **2**, 433.

to have been infected while attending to the needs of the tuberculous patients. The physicians who developed phthisis during the past ten years, have had histories of this disease before entering the service.

Similar statistics are available for hospitals in Germany and France, published by Aufrecht,[1] Freymuth,[2] Brunon,[3] Saugman,[4] and others, and brought together by the author[5] in a paper on hospital infection. Instructive data on the subject have been collected by Saugman from many sanatoriums in various countries. He finds that even among laryngologists, who are exposed to infection more than any other class, the morbidity and mortality from tuberculosis are less than would be expected. He concludes that tuberculosis is extremely rare among those who are engaged among consumptives; physicians and laryngologists who had been healthy before entering upon their duties, remain so. "It is not dangerous for healthy adults to be coughed at by patients suffering from pulmonary or laryngeal tuberculosis" concludes Saugman.

Such facts have been quoted to disprove the transmissibility of tuberculosis, but in the light of our present knowledge they merely prove that reinfection is impossible.

Marital Phthisis.—Bearing in mind the ease with which tuberculosis is transmitted to persons who have not been infected previously, it is to be expected that the vast majority of husbands of tuberculous wives, or healthy wives of tuberculous husbands should acquire the disease. This, we know, is the case with syphilis, in which the active disease is almost invariably transmitted to the unaffected consort, excepting when the latter has been infected before marriage. But for a long time it has been a mystery why phthisis in both husband and wife is very rare in spite of the fact that they probably come into more intimate contact than even father and child. Even in families in which most or all of the children are affected with tuberculosis it is exceedingly rare to find that both the mother and the father should be sick with the disease. Formerly this fact was used as a strong argument against the transmissibility of tuberculosis, but now we understand that it is due to the immunity acquired by an infection which has not been effective in producing phthisis.

For many years the writer[6] was physician to a charitable society, having under his care annually 800 to 1000 consumptives who lived in poverty and in want, in overcrowded tenements, having all opportunities to infect their consorts; in fact, most of the consumptives shared their beds with their healthy consorts. Still, very few cases were met in which tuberculosis was found in both the husband and the wife. Widows, whose husbands died from phthisis, were only rarely

[1] München. med. Wchnschr., 1908, **45**, 158.
[2] Beitr. z. Klin. d. Tuberk., 1911, **20**, 231.
[3] La tuberculose pulmonaire, Paris, 1913, p. 59.
[4] Ztschr. f. Tuberk., 1905, **6**, 125; 1907, **10**, 224.
[5] Am. Med., 1915, **21**, 607.
[6] Am. Jour. Med. Sci., 1917, **153**, 395.

seen to develop the disease. Among 170 couples in which one of the consorts was tuberculous, it was found that only in 2.5 per cent were both the husband and wife phthisical; this notwithstanding the fact that a large majority lived very closely together, even sharing the bed. It has been my impression when investigating this problem that if under such conditions infection has not taken place, it cannot occur in any other adults.

This experience is not unique. Mongour[1] found that among 440 married couples, in which one of the consorts was sick with tuberculosis, there were only 16 in which the partner was also phthisical, *i. e.*, 4 per cent. Thom[2] reports 402 couples with only 12, or 3 per cent, in which infection of the consort had taken place in all probability. I. Burney Yeo[3] found marital phthisis comparatively rare, basing his deductions on particulars collected of 1055 cases of consumption. He cites figures of J. R. Bartlett, Herman Weber, and others and concludes: "Taking these figures for what they are worth, it seems certain that the communication of consumption from wife to husband, even among the class in which the conditions of life favor to the utmost the communication of contagious disease, is very rare; while it would seem that communication from husband to wife is more frequent." Pope,[4] Pearson,[5] Elderton, and Goring have made careful statistical studies of this problem in England and arrive at the conclusion that the chances of tuberculosis occurring in both consorts are about the same as insanity, and a German writer has shown that cancer in both consorts is more apt to occur than phthisis. In a recent statistical study by Levy,[6] comprising 315 married couples which lived in poverty, 34 per cent sharing the bed, possible marital infection could be traced only in 2.8 per cent. He points out that when marital phthisis does occur, it is characterized by a favorable course of the disease in the secondary cases, and soon after the actively diseased partner is removed, the infected consort recovers his or her health. Haupt found among 1553 tuberculous couples that 106, or 7 per cent, were both affected. This being the highest percentage recorded, it is essential to remember that it is exactly the proportion in which humanity suffers from the disease.

In this connection it is important to mention a curious phenomenon, first mentioned by Petruschky[7] and which he named "mother immunity." A woman marrying a tuberculous husband begets children, most of whom either are sick with, or die from, tuberculosis, but she remains healthy. Gerald Webb[8] has observed the same condition,

[1] Cong. Intern. de la Tuberculose, Paris, 1905, **1**, 413.
[2] Ztschr. f. Tuberkulose, 1905, **7**, 12.
[3] British Med. Jour., June 17, 1882, p. 895.
[4] A Second Study of Statistics of Pulmonary Tuberculosis. Marital Infection. London, 1911.
[5] Tuberculosis, Heredity and Environment, London, 1912; The Fight against Tuberculosis and the Death Rate from Phthisis, London, 1911.
[6] Beitr. z. Klin. d. Tuberk., 1914, **32**, 147.
[7] Ergebnisse d. Immuntatsforschung, 1914, **1**, 189.
[8] Jour. of Laboratory and Clin. Med., 1916, **1**.

though he is inclined to consider this as only a relative and not a complete immunity of the mothers, because they react to tuberculin, and he even found one of them to be herself a "carrier." The present writer has had many women of this type under his care. Analogous conditions are seen in men, which may be called "father immunity." A man marries a wife who dies from tuberculosis; he again marries and his second wife succumbs to the same disease. I know of one who had three wives die from tuberculosis, while he remained healthy. The children are usually tuberculous, or die from this disease.

We have dwelt on these facts because they are very important points in phthisiogenesis: (1) Tuberculous infection can only occur once; and (2) phthisis develops only in persons who are, for one reason or another, constitutionally predisposed to the disease. Inasmuch as the non-phthisical consort has already been infected with tubercle bacilli during childhood, new opportunities for reinfection by cohabitation with a consumptive consort are of no avail to produce phthisis. It is his or her constitution that determines whether consumption will develop, and not the opportunities for reinfection.

Clinical Evidence of Immunity Acquired by Tuberculous Infection.—Recent investigations have shown that a tuberculous bacteremia is not uncommon (see p. 284) in patients suffering from any of the forms of tuberculous disease, yet they do not manifest symptoms of acute miliary tuberculosis, as would be expected *a priori*. Similar immunity phenomena have been observed in syphilis and malaria, and Morgenroth[1] has shown this to be the case in chronic infections with hemolytic streptococci. Whether it is a form of "depression immunity," as Morgenroth suggests, or to some other cause, remains to be decided by further investigations, but there seems to be no doubt that mutual immunization of the parasite and host does occur in tuberculous infection.

A number of clinical facts, hitherto obscure, can be rationally explained by this acquired immunity of the tuberculous to the virus of tuberculosis, and they confirm the assumption that certain experimental data obtained in animals hold good for man. Thus, despite the fact that so much sputum containing tubercle bacilli passes through the throat, mouth, lips, etc., tuberculosis of the mucous membranes and the cervical glands in adults is exceedingly rare. Patients with extensive tuberculous lesions of the lungs, expectorating myriads of tubercle bacilli, often have their teeth extracted or the tonsils removed by operation, yet no local tuberculous infection occurs, though tubercle-laden sputum surely remains on the open wounds.

Scrofulous children have been noted by physicians for many generations to display great resistance against the development of active tuberculous disease of the lungs in later life; many even speak of immunity. The same is the case with other extrathoracic tuberculous

[1] Deutsch. med. Wchnschr., 1920, **46**, 337.

lesions. As will be shown later on (see Chapter XXX), persons with active tuberculosis of the glands, bones, joints, skin, etc., only rarely develop pulmonary phthisis. Mayo[1] points out that in Minnesota, where surgical tuberculosis is rife, phthisis is uncommon, and this has been observed to be the case in many other places. A large proportion of the population of Colorado is known to be descended from persons who emigrated to that State because of tuberculous disease. It has been observed, and recently confirmed by Whitney,[2] that Coloradoans display a striking immunity against active tuberculosis. While the climate may be credited, still it does not explain this phenomenon completely. Natural selection, eliminating the susceptible stock, favors the propagation of those who have strong resistance. Their survival, despite the pathological stock, indicates that they possess unusual resistance, and that their descendants share in it. Clinical experience teaches that the prognosis in tuberculous patients is much better, and the course of the disease less stormy, in those who have descended from tuberculous parentage, or who were "scrofulous" during childhood (see Chapter XXX).

The immunity of the tuberculous to tuberculosis is thus confirmed by a variety of clinical facts which can hardly be explained otherwise.

Tuberculosis on "Virgin Soil" in Human Beings.—In contrast with the immunity displayed by infected human beings against reinfection with tubercle bacilli, those free from the virus are very susceptible, and when infected the clinical phenomena are the same as those observed in experimental tuberculosis in animals. Bearing in mind that newborn infants are free from tuberculosis, no matter from what stock they are descended, it is suggestive along the lines of our inquiry that when infected, the resulting disease runs an acute and progressive course, just as is the case in experimental tuberculosis in guinea-pigs and rabbits. For obvious reasons, no experiments of this kind have been made on infants. But there have been observed inoculations of tubercle in newborn infants among Jews when the wound made during ritual circumcision was infected by a tuberculous operator. The infant promptly becomes sick with tuberculosis, and the disease runs an acute and progressive course, the regional lymph glands being implicated. This is a drastic contrast to the mildness of tuberculous inoculation infections in adults, as is seen in the "butcher's wart," the "pathologist's wart," etc. Moreover, there are available certain observations which tend to show conclusively that adult human beings cannot be experimentally infected with tubercle bacilli. In February, 1900, Felix Klemperer[3] injected subcutaneously bovine bacilli into his own arm. Ten months later he excised the indurated subcutaneous cellular tissue at the site of the injection and microscopic examination showed well-organized granulation tissue with giant cells, but no caseation.

[1] Jour. Am. Med. Assn., 1905, **44**, 1156.
[2] Colorado Medicine, 1919, **16**, 268.
[3] Ztschr. f. klin. Med., 1905, **56**, 241.

No tubercle bacilli could be discovered, showing that specific disease was probably absent, and the tissue changes were, at any rate, not characteristic of tuberculosis. Another physician, who had been tuberculous for fourteen years, also submitted to similar injections of bovine bacilli. In this experiment the individual was given fourteen injections without producing any results. Four other tuberculous patients were injected with tuberculous lymphatic tissue from guinea-pigs. A total number of thirty-nine injections of bovine bacilli were administered to these four patients. The local effects were slight. Four times abscesses were produced which, however, healed sooner or later. General constitutional effects were not observed in any case; the patients stated that they felt better, and they gained in weight during the experimental treatment. Klemperer concluded that, within certain limits, there appears no doubt that subcutaneous injections of bovine bacilli are harmless to tuberculous individuals. Baumgarten[1] performed similar experiments on cancerous patients with the same results.

It is important to mention that experimental infections of physicians have, almost without exception, proved harmless. Thus, Alfred Möller[2] was infected intravenously with tubercle bacilli. He did not develop any acute disease, excepting that he lost in weight for a few months and then recuperated without showing any symptoms of tuberculosis. Baldwin and Gardner also mention that Garnault injected virulent bovine bacilli into his own forearm with no harmful results. Ritter and Vehling[3] report accidental inhalation without harm of millions of dry, virulent tubercle bacilli by Hans Much and his coworkers. "It is difficult to avoid such accidents," say Baldwin and Gardner,[4] "in research laboratories where dry material is manipulated, yet to our knowledge no authentic instance of their causing pulmonary tuberculosis has been reported."

Phthisis as a Manifestation of Immunity.—From the experimental and clinical data arrayed above, it is clear that neither infection with tubercle bacilli, nor predisposition, whatever this may consist in, is alone capable of producing tuberculous disease. To each one who becomes phthisical, there are many who have been infected with tubercle bacilli and remained healthy in the clinical sense. Indeed, spontaneous infection acquired during childhood appears to render the body immune against further and renewed exogenous infection with the same virus.

It is also clear that *chronic phthisis occurs only in individuals who have been infected with tuberculosis during childhood*, but have remained healthy until adolescence. In other words, *phthisis occurs only in persons who have been immunized by an earlier infection*. In fact, it is in itself a manifestation of immunity, otherwise the patient would succumb to

[1] Berl. klin. Wchnschr., 1901, p. 894; 1905, p. 1329.
[2] Ztschr. f. Tuberk., 1904, **5**, 211.
[3] Berl. klin. Wchnschr., 1909, **56**, 1914.
[4] Am. Rev. Tubercul., 1921, **5**, 436.

acute general miliary tuberculosis, as do those who have not been immunized by earlier mild infection. This immunity is apparently sufficient to protect the individual under ordinary circumstances, but under certain conditions it may fail, and the person may be reinfected either from without, the tubercle bacilli being so ubiquitous that we can hardly escape them; or from within, by the proliferation of the bacilli that have been harbored in "healed" or quiescent foci, through metastasis.

Acquired immunity in contagious diseases is hardly ever absolute—it is only relative, sufficient for the ordinary conditions of ife, and failing during emergencies. The same appears to be true of the immunity acquired during childhood by infection with tubercle bacilli. It protects the average person against exogenous reinfection with tubercle bacilli, and moderate failure of immunity permitting reinfection does not result in general tuberculosis, but only in phthisis—the most vulnerable organ in the body, the lung, succumbs, while the others are still more or less protected.

Immunity through Bovine Infection.—Some authors have been inclined to attribute the immunity observed in most adults to infection during childhood with the bovine type of bacilli which protects the individual against superinfection with bacilli of the human type. Clive Riviere[1] even advocates the immunization of humanity along these lines. He says that "until human sources of infection can be practically eliminated, or artificial immunization becomes an accomplished fact, infection with the bovine bacillus through the use of a well-mixed milk remains our best ally in the campaign against tuberculosis." We have seen already that bovine infection is fatal only in exceedingly rare instances. That it may protect against infection with the human type of bacillus is made highly probable by the rarity of phthisis in surgical tuberculosis. "Very significant in this respect also are the figures of McNeil for Edinburgh where, as shown by Fraser and Philip Mitchell, tuberculosis of bovine origin is particularly rife. Comparing Edinburgh with Vienna, he finds the incidence of tuberculous infection higher in the former for children up to the age of four years, and this in itself is highly suggestive of milk infection; but the valuable comment on this is the fact that the mortality from phthisis in Vienna is nearly three times as high as that for Edinburgh. Indeed, the high incidence of abdominal tuberculosis and the low mortality from phthisis are characteristic of Great Britain as a whole when compared with other civilized countries of Europe, and this may well bear the interpretation that it is the early bovine infection which protects against the inroads of pulmonary tuberculosis caused by the human strain of tubercle bacillus." Brownlee[2] also found that in England phthisis is less common in those districts in which there is a large

[1] British Jour. of Tuberc., 1914, **8**, 83.

[2] An Investigation into the Epidemiology of Phthisis in Great Britain and Ireland, Part III, London, 1920, p. 55.

number of deaths from tuberculosis in children, and also in the counties from which a large amount of milk infected with tubercle is sent to London. On the other hand, Bushnell[1] is inclined to think that children become immunized against infection with bovine bacilli, which they often ingest with milk, through previous contact with human tubercle bacilli.

Failure of Immunity.—Obviously the evolution of phthisis does not depend alone on the intensity of the infection during childhood. The character of the soil invaded by the bacilli is perhaps more important. Some succumb to hematogenous tuberculosis as a result of a mild infection, harmless to the average individual, which indicates that predisposition was a stronger factor. In what this predisposition consists we are in the dark, as has been shown in Chapter IV, in which most of the facts and fancies of this problem have been discussed in detail.

Failure of immunity may be due to various complex biochemical changes in the body with which we are unacquainted at the present state of our knowledge. This is seen in children who have been infected but who thrive in spite of it, until an attack of measles, whooping-cough, etc., which is accompanied by a failure in allergy, as is evident from the negative outcome of the cutaneous tuberculin test during the active stage of the disease, flares up the latent tuberculous focus and tuberculous bronchopneumonia results. Other febrile diseases may act in the same manner, but we do not as yet know the exact nature and effects of these biochemical changes in the body following contagious disease.

The nature of predisposition is the stumbling-block of the theories of phthisiogenesis. Clinical, demographic, and experimental observations have not cleared up these important problems. It appears that no single predisposing factor, nor a combination of several factors, will fit most cases. As has been pointed out by Martius,[2] the predisposition of the individual is, after all, not a specific entity, which is possesed by those who are attacked by phthisis, and lacks in those who escape the disease despite infection. It appears to be a complex affair: In each individual case there are a number of anatomical and physiological factors which may each alone, or several in combination, decide, under certain conditions whether the person is to become phthisical and even these factors are subject to great oscillations, and may combine differently under different conditions. From this point of view everybody is predisposed to tuberculosis, but there are many important differences in the resisting powers of different individual persons which depend on the number, intensity, and accidental combinations of the various predisposing factors which, by themselves, are influenced by certain vital, biological oscillations occurring during the lifetime of the individual. We thus have gradations of predisposition from the strongest degree of vulnerability to the highest degree of immunity.

[1] Epidemiology of Tuberculosis, New York, 1920, p. 180.
[2] In Brauer, Schröder, and Blumenfeld's Handbuch d. Tuberkulose, **1**, 395.

The various clinical forms of tuberculosis, acute, subacute, chronic, fibroid, etc., may thus be explained to a certain extent.

Endogenous and Exogenous Reinfection.—Considering phthisis as a disease which develops only in an organism that has been immunized by an earlier infection which has left a latent or "healed" tuberculous focus in some part of the body, the problem arises whether the flaring up of the local lesion in the lung is caused by a new infection from without, by the invasion of new bacilli, or from within by metastatic migration of bacilli which have been kept dormant for years, until the immunity they conferred fails for some reason.

Experimental findings on this point are somewhat conflicting. Orth and Rabinowitsch[1] have found that when guinea-pigs are infected with very small doses of mildly virulent tubercle bacilli which cause only local tuberculous changes, the effect produced is that a second infection with virulent human bacilli does not cause the usual generalized tuberculosis, but pulmonary tuberculosis results, bearing some analogy to pulmonary tuberculosis in human beings. In rabbits, which react to human bacilli in a manner similar to that of man more than guinea-pigs, they produced in this manner chronic tuberculous lesions in the lungs. Hamburger, Bartel, Levy, Hugo Selter, and others have confirmed these findings. This would indicate that phthisis is due to exogenous superinfection.

That the outbreak of phthisis is due to autogenous, or metastatic, reinfection has been maintained by Behring, according to whom the primary infection takes place through the gastro-intestinal tract during childhood, the bacilli remaining latent until stirred into activity by some exciting cause. But if this were the case we should expect that pulmonary tuberculosis due to bovine bacilli would be very frequent, considering that at least 10 per cent of infections during childhood are caused by this type of microörganisms. As it is, there have been reported very few cases of phthisis in which the bovine bacillus was found exclusively. We have already mentioned that it has been suggested that those infected with bovine bacilli are immune against human bacilli, and they are the ones who escape phthisis despite tuberculous infection, but this would have to be proved.

Römer and Much maintain that their investigations lead them to the conclusion that reinfection is always endogenous, or metastatic, from existing tuberculous foci within the body. "We know," says Much,[2] "that a tuberculous organism is not susceptible to, in fact it is immune against, superinfection from without. We must also admit that when an organism is infected during childhood it passes through a precarious crisis, but it may survive this first infection and remain endowed with immunity. But during adolescence, when great demands are made upon the vital forces, the body may be overwhelmed by the bacilli and the most vulnerable organ in the body—the lung—succumbs:

[1] Drei Vorträge über Tuberkulose, Berlin, 1913.

[2] In Brauer, Schröder, and Blumenfeld's Handbuch d. Tuberkulose, **1**, 247.

thus phthisis results. One who hesitates in accepting these ideas of reinfection from within should only compare phthisis with syphilis."

There are analogous conditions known in pathology showing that an organism may harbor virulent bacilli without any harm to itself. Thus, the "carriers" of typhoid, diphtheria, meningococci, pneumococci, and other bacilli may go around for years without showing any symptoms of disease, although they are a constant danger to others. But Texas fever illustrates this point even better. Cattle which survive an attack remain with the living virus within their bodies, but are immune against new infections, so that they may remain in infected pastures without any danger to themselves. But should they suffer from any secondary derangement, they may, as a result, experience an acute exacerbation of the process owing to sudden proliferation of the virus which has been dormant for a long time within their bodies.

There are similar clinical phenomena in man. It is known that infection with the malarial parasite protects against further infection with the same parasite from external sources, and for this reason indigenous adult individuals in malarial districts are immune to malaria, as was shown by Koch. In some cases there occurs further infection in later years, and the result is a cachexia, a sort of malarial phthisis. But in such cases the initial infection must have been an especially strong and severe one. In syphilis this is even illustrated to better advantage. Superinfections are very difficult, usually impossible; the integuments and mucous membranes cease to react to the syphilitic virus introduced from without while they are susceptible to their action from within. John A. Fordyce,[1] in a review of this subject, cites several other examples: "Levaditi has demonstrated that animals suffering with spirillary infection are immune to a new inoculation. Their serum has a high antibody content, but the blood still harbors parasites and is capable of producing a fresh infection in healthy animals. So with the serum of guinea-pigs inoculated with Nagana or Surra trypanosomes. This is trypanocidal for these organisms *in vitro*, but *in vivo* they have acquired an insensibility to the trypanolytic antibodies, for the blood and tissues of the animals still contain parasites. The same is true of human subjects suffering from sleeping sickness in whose serum trypanolytic, agglutinating, and other protective bodies have been demonstrated. Carrying the analogy to syphilis we find that an individual may harbor spirochetes for forty to fifty years, while his skin and mucous membranes exhibit an insusceptibility to reinoculation under natural exposure. However, as soon as he is freed from his infection he is again in as susceptible a state as he was prior to his first attack."

We have shown that healed tuberculous lesions contain living and virulent tubercle bacilli; in fact, even calcified foci contain them. It has even been questioned whether once infected with tubercle bacilli,

[1] Am. Jour. Med. Sci., 1915, **149**, 761.

the virus is ever absent from the body. And for this reason we may look upon phthisis as produced by endogenous reinfection. Thus, according to Römer, phthisis is an acute or subacute exacerbation of a latent or quiescent lesion in the lungs acquired by massive infection during childhood, the bacilli remaining dormant for years, but when the immunity which they conferred failed, owing to some intercurrent disease, the lesion in the lungs flared up. That the specific immunity is not altogether lacking even under these circumstances is evident from the fact that the lesion remains localized for a long time in the most vulnerable of organs—the lungs. Phthisis is thus proof of immunity against tuberculosis. General miliary tuberculosis does not develop easily in the vast majority of individuals who have been immunized by previous infections with tubercle bacilli.

Immunity of Adults.—The question why adults are not immunized by mild primary infections, as children are, has not been explained satisfactorily. We have already mentioned that adults hailing from countries where tuberculosis is unknown, and where they could not have been infected during childhood because of the lack of tubercle bacilli, upon coming into cities, and in contact with tubercle-laden surroundings—subjected to primary tuberculous infection—soon succumb to the acute forms of phthisis, like infants or guinea-pigs. Cobbett[1] is inclined to attribute it to the cessation of the strain made upon the constitution by bodily growth. Be that as it may, he thinks that we may conclude that infection with tubercle bacilli, though it does not entirely cease when adult age is reached, is nevertheless, like infection with most other diseases, less easily acquired then than in childhood and adolescence. Much attempted to explain it by saying that either the organism of the child alone is capable of evolving a sufficient quantity of immune bodies, or we must assume that an adult person, coming from an environment free from tuberculosis to one which is tubercle-laden, freely going around among people among whom there are many bacilli carriers, is soon subjected to massive infection against which he does not possess sufficient powers of resistance. On the other hand, the sheltered infant does not roam around among various people during the first years of its life and comes in contact with only a few bacilli, so long as there is no active case of tuberculosis at home. I may add that the suggestion made above to the effect that the immunization of humanity during childhood may be accomplished by the bovine type of bacillus, which is not so virulent as the human type, may be responsible for this salutary condition. But this problem has not yet been worked out.

Summary.—At the present state of our knowledge of tuberculous infection and immunity, particularly as regards chronic phthisis, the following conclusions appear justified:

In civilized communities nearly all adults have been infected, though not all have acquired disease by virtue of this infection.

[1] Practitioner, 1918, **100**, 404.

Infection occurs in nearly all cases during childhood, the bacilli remaining latent within the body until some exciting cause reactivates them, or the natural resistance is reduced, and tuberculous disease results by endogenous reinfection.

Infection during childhood, so long as it is not acute and fatal soon after the bacilli have entered the body, endows the organism with heightened resistance against renewed endogenous and exogenous infection with tubercle bacilli. The immunity thus produced is, in most persons, ample to protect them against exogenous or endogenous reinfection with tubercle bacilli during the rest of life.

When, for any reason, this immunity fails and the bacilli within the body are permitted to proliferate, metastatic reinfection may occur, new tuberculous foci develop, and clinical phenomena of tuberculous disease make their appearance. Experience tends to show that such metastatic reinfections mostly occur in individuals who were subjected to massive infections during childhood.

Phthisis is thus a manifestation of immunity against exogenous and endogenous reinfection and superinfection with tubercle bacilli. When for any reason this immunity fails, no acute miliary tuberculosis develops, as is the case in massive primary infections, but only a local lesion results, the most vulnerable organ in the body—the lung—succumbs.

CHAPTER VI.

PATHOLOGY AND MORBID ANATOMY.

THE TUBERCLE.

TUBERCLE bacilli settling on susceptible soil offering suitable conditions for their growth induce a specific proliferation of the fixed elements of connective tissue, capillary endothelial, and probably also of the epithelial cells of the air vesicles, as well as an invasion of wandering cells. Acting as irritants, and injuring the cells and the intercellular substances, they induce a productive imflammation resulting in the formation of a nodule, the specific granuloma termed *tubercle* by Laennec.

This unit of the characteristic tuberculous process, *the tubercle*, is best studied in acute miliary tuberculosis, where tubercles of all ages—corresponding to successive invasions of bacilli into the bloodstream—are usually found. Throughout the lungs are scattered small, hard nodules. The younger ones are gray and translucent, the older, yellowish white and opaque. The transparent tubercles are smaller than millet seeds, while the opaque ones are larger, as a rule. They are almost always larger and more numerous in the upper parts of the lungs where they grow better and more rapidly, which is perhaps correlated with the inferior blood supply of the upper lobes. Individual tubercles are too small to be seen clearly with the naked eye, and what we really see is for the most part fusions of several, *conglomerate tubercles*.

Microscopically, the primitive tubercle presents a characteristic structure (Figs. 21, 22 and 23) of a fairly well-circumscribed conglomeration of cells. Primarily it is avascular; with the growth of the cells, the lymphatics and bloodvessels in its neighborhood are compressed and obliterated. In the typical young tubercle we see a multinuclear element called the *giant cell*, around which are cells which resemble epithelial cells in appearance and arrangement, and hence are called *epithelioid cells* (Fig. 23). At the periphery there is a stockade of lymphocytes.

The Giant Cell.—The giant cell is most typically found near the center of the tubercle. But there may be several giant cells in a tubercle or, more rarely, there may be none. Exceptionally, particularly in conglomerate tubercles the giant cell or cells may be located toward the periphery. The giant cell is a multinuclear element with a stroma of fatty degenerated, or even necrotic protoplasm. It may contain as many as one hundred oval, spindle-shaped nuclei arranged concen-

trically like a crescent. The tubercle bacilli are mainly located in the giant cells (Fig. 22), where they may be seen singly or in clusters, usually at the inner side of the nuclei, or between the latter. They are, however, lacking in the center of the protoplasm of mature giant cells; probably the process of necrobiosis affects the bacilli as well as the body of the cell. In some tubercles the nuclei are located at the two poles (Langhaus' type of giant cell), and occasionally there may be nuclei in the center. The cytoplasm in which the nuclei lay appears either quite homogeneous, or but faintly granular. Quite long streams of it can usually be traced between the neighboring epithelioid cells. In

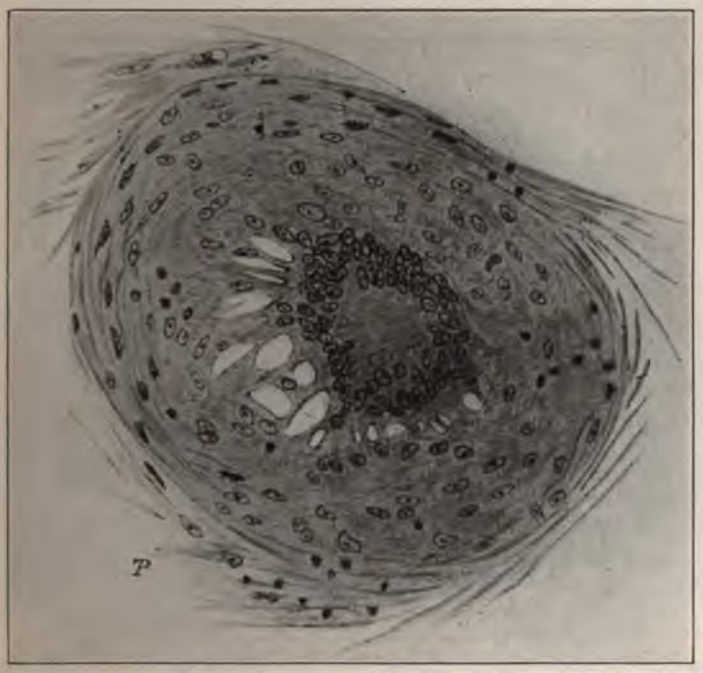


FIG. 21.—Microscopic tubercle. (Tendeloo.)

thin sections, a fine network, the reticulum, is seen. The filaments are derived partly from extravasated fibrin, partly from curled fibrils of connective tissue, and partly from long, branching, interlacing processes of the cells, especially the giant cells, which have been described as looking like spider's feet, and also from newly formed connective-tissue cells.

The origin of the giant cells has been a debated subject. Some, like Weigert and Baumgarten, consider them the results of karyokinetic changes of the nuclei which retain the capacity for division, while the protoplasm, owing to the necrobiotic effect of the tubercle bacilli, does not divide into separate cells. In fact it is quite common to find

in tuberculous foci cells with degenerated protoplasm, while the nuclei show an increased chromatin content. From this point of view the giant cell is a degenerative phenomenon. On the other hand, Metchnikoff sees in the giant cells a manifestation of phagocytosis. They are macrophages, or large active phagocytes, produced by the fusion of many epithelial cells with the object of fighting the invading enemy, the tubercle bacilli, with united forces. The part of the giant cell which has no nuclei is usually dead, because of the noxious effects of the tubercle bacilli. It is thus seen that the question whether the giant cells are derived from the fixed tissue elements (endothelial and connective-tissue cells, etc.), or result from the fusion of a number of cells,

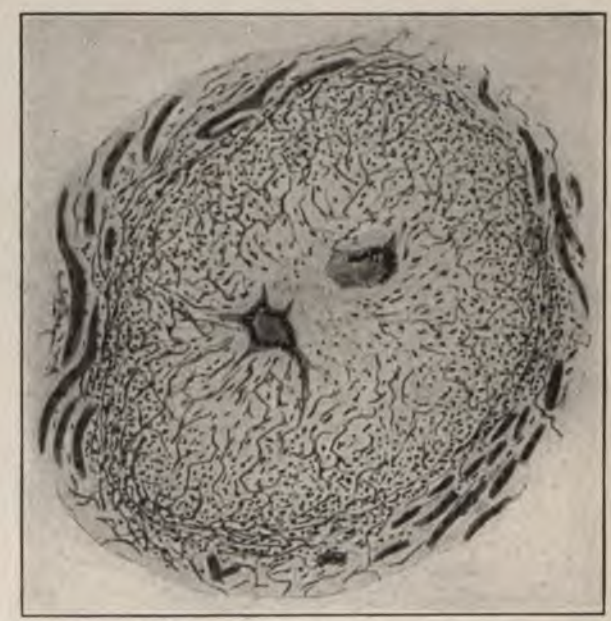


FIG. 22.—Tubercle, slightly enlarged, two giant cells in the center. (Ribbert.)

or repeated division of cell nuclei without accompanying splitting of the cell body, has not been solved. If the latter is true, the division must be amitotic, for mitotic figures are not seen in the nuclei of the giant cell.

Tubercle bacilli are mainly found in the giant cells, as has already been mentioned, and also in the epithelioid cells, while in the intercellular substance they are rarely noted. In the caseous parts of the tubercle, bacilli are found at the periphery, while they are never seen in the center. In the caseated giant cells they are found only in the parts which have retained their staining property.

The Epithelioid Cells.—The epithelioid cells are scanty in some tubercles, because they are composed mainly of lymphoid cells (lymphoid tubercles), but more often the epithelioid cells predominate

(epithelioid tubercles). These cells are rounded, or somewhat elongated elements, whose cell body stains but palely in the ordinary hematoxylin-eosin preparation, and is not very clearly outlined. The nucleus usually contains but little granular chromatin. As is the case with the giant cells, there is no unanimity as to the origin of these epithelioid cells. Maximow thought that they are derived from the lymphocytes. Baumgarten attributed them to the proliferation of the fixed tissue cells, while others see in them results of wandering cells coming from the blood stream. In a recent study of experimental tubercles, N. C. Foot[1] found confirmation of the suggestion that the epithelioid cells are derived from the vascular endothelium of the lesion. They, accord-

FIG. 23.—Part of a tubercle very much enlarged; around the large multinuclear giant cell there are numerous epithelioid cells. (Ribbert.)

ing to Foot, not only phagocyte tubercle bacilli, but carry them into the tissues, for example into lymph nodes by way of the lymphatics, or into other lung lobules by way of the air passages in which they are readily demonstrable. It is probable that the epithelioid cells may arise from the several varieties of fixed cells, depending on the location of the tubercle, such as capillary endothelium, ordinary connective-tissue cells, the Kupfer cells of the liver, the mesothelial lining cells of the serous sacs, etc. Still, it is possible that in some cases the epithelioid cells may arise from wandering cells.

It has been demonstrated that the epithelioid and lymphoid cells

[1] Jour. Exper. Med., 1920, **32**, 513, 533.

lie in a fine reticulum which is partly, but apparently not wholly composed of fibrin. Also, here and there may be seen some remnants of the more resistant of the structures previously present, such as fragments of elastic tissue, etc.

Histogenesis of the Tubercle.—The origin of the tubercle has been a debated subject for a long time, but the detailed studies of Baumgarten[1] have thrown much light on it. It has been noted that when tubercle bacilli are arrested in a small capillary, or on the wall of a terminal bronchiole, etc., the first reaction is not a dilatation of the neighboring vessels, exudation of numerous leukocytes, etc., as is the case in ordinary acute inflammatory processes, but rather the proliferation of the neighboring fixed tissue cells (endothelial cells, connective-tissue cells, etc.), which surround the bacilli. As was already stated

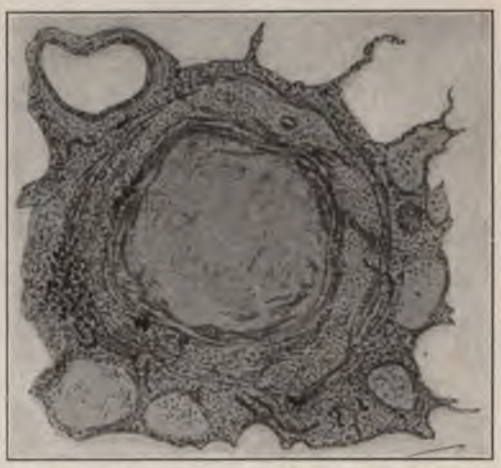


FIG. 24.—Cross-section of tuberculous bronchus. The lumen of the bronchus is completely filled with muddy but quite homogeneous caseous matter and the mucous membrane has completely vanished. The rest of the bronchial wall is very rich in cells, and thickened. The thickening extends far into the neighboring alveoli. (Ribbert.)

above, the proliferation of these fixed tissue cells results in the formation of the epithelioid cells, and several of them may fuse around the bacilli to form a giant cell (in the formation of which cell division may also play an important part). Some of the cells are destroyed by the toxic action of the bacilli, and as a result some wandering cells are then attracted. At first these are the polynuclear, but these are soon replaced by lymphocytes, which form the peripheral part of the tubercle.

Caseation.—The tuberculous follicles are therefore avascular neoformations, and their vitality is not durable. No new bloodvessels are formed in them, as is the case with most other new growths. They are usually located in the alveolar framework, whence they compress

[1] Ztschr. f. klin. Med., 1884, **9**, 93; 1885, **10**, 24.

PLATE II

FIG. 1

FIG. 2

Fig. 1.—*C*, cavity in the pulmonary apex; *F*, interlobar fissure. To the left of the cavity are seen peribronchial nodules. Lower parts are extensively caseated.

Fig. 2.—*C*, small caseous focus in the upper part of the apex; *B*, bronchus with caseated wall. The rest of the parenchyma is of normal air content, but anthracotic and showing black pigmentation. (Albert Fraenkel.)

the neighboring alveoli and finally obliterate them, and partly in the smallest lymph vessels, *i. e.*, along the walls of the smallest arterioles and bronchioles. In the arterioles a tuberculous obliterative endarteritis is formed and this alone, or in conjunction with thrombotic phenomena, leads to occlusion of the vessel. In the small bronchioles caseous bronchitis may result, which may, however, arise primarily and lead to peribronchial tuberculosis secondarily. The bronchi become permanently plugged by their own secretions and by the irritative proliferation of their epithelium. The tuberculous growth finally compresses and destroys even the elastic fibers, so that in the center of the nodule there are only fragments of these tissues and often not even these, and air is completely excluded.

The necrotic tissue is thus converted into a whitish or muddy, yellowish opaque mass; dry, often fragile, at times soft, or even viscous in consistency. It has the appearance of dry or soft cheese. Microscopically, the cells are found to have undergone fatty changes and ultimately death occurs by the process which Weigert called coagulation necrosis, the cells are converted into a structureless mass of detritus which refuses to stain. At times, we make out between the remnants of the cells a filament, consisting of a fine network of granular fibrin, or true hyaline fibrin, the so-called "fibrinoid reticulum." Finally, a stage is reached when the débris of cells and fibrin become a homogeneously granular mass in which no structure is seen at all. This is true caseous matter.

Some have suggested that tuberculous toxins are specifically effective in causing necrobiosis of the affected cells, but this has not been proved. It must be emphasized that desquamation of epithelial cells, necrosis, and caseation are not specific tuberculous changes. They are found also in various degrees of intensity in several other inflammatory processes in the lungs. Necrosis, especially coagulation necrosis, is also found in diphtheritic inflammation of mucous membrane, etc., and caseation in gummatous changes. The caseous gummatous nodule is often difficult to differentiate from the tuberculous.

Calcification.—The caseous matter may become surrounded by a layer of connective tissue—encapsulated—and then, by the exclusion of water, it becomes inspissated and much reduced in size. In time small granules of calcium are gradually deposited until it becomes altogether calcified. Small calcified granules may coalesce into larger concretions, until finally they are converted into a dry, solid, jagged, or fragile concretion which looks very much like chalk. These concretions often contain virulent bacilli. In general, it can be stated that they are never dissolved or absorbed by autolysis, as is the case with other dead matter in the tissues. But caseous matter may be gradually permeated by fibrinous tissue and finally converted into a solid fibrous scar.

Softening.—Very often the tubercle, instead of calcifying or undergoing fibrosis, softens as a result of the action of proteolytic enzymes

with which we are yet unacquainted. In this case there develops a puriform, thin liquid, without any pus cells but containing bits of cheesy matter, which is known as puriform liquefaction and "tuberculous pus." In other cases real pus is formed, or a mixture of both liquids, which is also known as tuberculous pus.

Sclerosis.—But the tubercle is not always destined to necrosis, caseous degeneration, calcification, or liquefaction. In most cases in which phthisis does not develop at all, or is checked in its progress and healing finally results, the nodule is converted into fibrous scar tissue through the agency of the proliferating connective-tissue cells. These connective-tissue cells are derived from two sources: From the

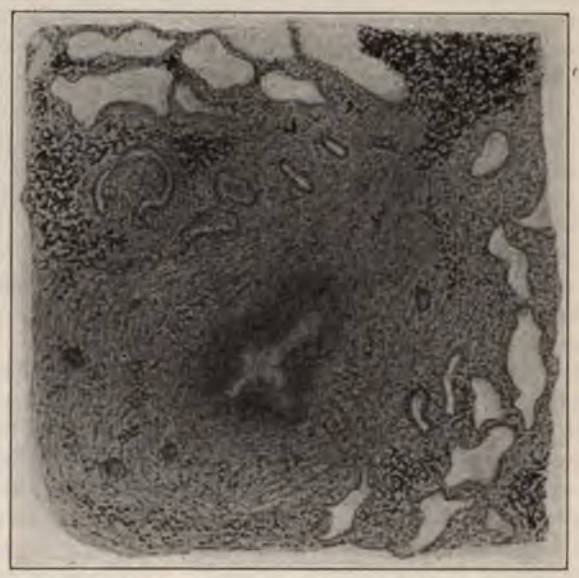


FIG. 25—Indurated nodule in pulmonary tuberculosis. The solid nodule has a dark, caseous center with irregular lacunæ. It consists of coarse connective-tissue fibers in which carbon particles are deposited in some places. A giant cell is seen in the middle and to the right; three others are seen to the left. (Ribbert.)

cells in the neighborhood of the tubercle, and from the tubercle itself. While making autopsies on persons who died from any cause pathologists have found that a large proportion have scars in their lungs and pleuræ, thus showing that an enormous number of persons have had tuberculosis which healed spontaneously. These healed or dormant lesions are responsible for the large number of persons obviously non-tuberculous, yet responding to the tuberculin test.

The fate of the tubercle depends on the intensity of the two processes of connective-tissue proliferation or sclerosis, and of caseation. In fact, the clinical course of the disease is mainly influenced by their relative intensity, the former being reparative, and the latter destruc-

tive. If the exudative process predominates and progresses with rapidity, the tuberculous focus increases in size and clinical significance; but when the proliferative process predominates, the inflammation proceeds slowly, and may even terminate in a cure through sclerosis. In chronic phthisis the two processes usually go hand-in-hand; the reparative, manifesting itself by the proliferation of connective-tissue cells, is seen at the periphery of the tubercle, while the center caseates. Pathologists then speak of fibrocaseous phthisis. In conglomerate tubercles the central foci may caseate, while those at the periphery are healing by sclerosis, and thus surround the lesion and prevent its progress by encapsulation of the cheesy center which finally calcifies, as was already shown.

Tubercles in the Lung.—Gross Appearances.—In the vast majority of cases the tuberculous lung is found at autopsy to be adherent to the inner surface of the thoracic wall, at least the affected apex is found densely adherent. Very frequently the pleural sheets are so thick and dense that the lung cannot be removed from the thorax with ease, but must be torn forcibly, or cut away. In some cases the entire pleura is thick, and the pleural cavity is completely obliterated. The apical and diaphragmatic pleural sheets are, however, the parts most often thus affected.

The external appearance of the affected lobe in chronic phthisis is irregular, deformed, or puckered, and of comparatively solid consistency. Frequently the surface of the lung is found studded with small pleural or subpleural tuberculous nodules; the interlobar fissure below the lobe in which the main lesion is located is usually obliterated by adhesions. The intrathoracic lymphatic glands, the hilus, mediastinal, and tracheobronchial are enlarged, hard and often dark because of anthracosis. On section these glands may be found in various stages of tuberculous degeneration, caseous, fibroid, or calcified. The first foci usually take root in the neighborhood of the apices and may remain there exclusively for a long time; in progressive cases, they extend by the production of new nodules. They usually consist in a combination of both productive inflammation in the form of nodule formation and a pneumonic process. The first tubercles occur as single and isolated nodules, or groups, around the bronchi and the bronchioles, and at times also around the walls of the larger bronchi and the bloodvessels—peribronchial and perivascular tubercles. Varying with the intensity of the affection and the resistance of the individual, the nodules enlarge and extend slowly or rapidly and new ones appear around them. Large conglomerations of tubercles may thus be formed. In progressive cases the tubercles do not remain separated for a long time, but by fusion of many the focus enlarges and extends. The central nodules sooner or later begin to disintegrate and are converted into caseous matter. But in most cases a sclerotic process may be detected which limits its progress, excepting in the very acute types of the disease.

On section the gross appearance of the typical tuberculous lesion in the lung presents a very variegated picture. In fact, there are hardly two cases which look alike. The scar tissue surrounding the cheesy centers, or insinuating itself within many caseous and softened areas, is a very strong substance made up of thick fibers and can be recognized by its color. It is dark because particles of carbon derived from the inspired air are deposited in it, and they cannot be expelled by expectoration because of their inability to reach the bronchial glands owing to the fact that the lymph channels are occluded or obliterated. It is therefore more or less dark gray, or even black in color, which contrasts distinctly with the various other colors of the lungs. The distribution of scar tissue is variable. In some cases it is mainly in the center of a group of tubercles, or it surrounds the caseated masses with extensive processes. A black, round or radiating scar may enclose a nodule the size of a pea or even larger, or several nodules. The cheesy matter is dry, and when old, calcified. This is very often found at the apex of clinically healed pulmonary tuberculosis.

Later the caseous matter softens and, when the degenerative process extends, reaching and implicating the bronchial mucous membrane, the softened débris may break through the alveoli or the bronchi. But in most cases sclerosis prevents the spread of the lesion, and even encapsulates it with a more or less dense fibrous shell. Within the capsule the caseous matter dries up and finally calcifies, and it is stated that small foci may even be absorbed, though this is doubtful.

There has been quite some discussion as to the origin of ulcerations on the surface of the bronchial mucous membrane and in the parenchyma of the lung. Some have considered these as the points at which the infecting bacilli have entered with the inspired air and set up the disease; that these ulcerations represent the primary tuberculous lesions. As far back as 1876 Parrot pointed out that in all cases of tracheobronchial adenitis such a primary lesion may be found in the lung if carefully searched for. This is known among French pathologists as *la loi de Parrot*, Parrot's law. G. Kuss[1] has confirmed Parrot's findings on extensive autopsy material, and more recently Anton Ghon[2] has found the same condition while doing numerous autopsies. French authors refer to these primary lesions as *chancres tuberculeux*, and the enlarged regional glands which are almost invariably found, as *bubons d'emblée*.

Others maintain that there are many cases of tracheobronchial adenopathy in which such a primary lesion in the bronchioles or pulmonary parenchyma cannot be discovered at the autopsy. It is also shown that even when found it should not be concluded in all cases that this ulceration represents the point of entry of the bacilli. It may be due to extension of the peribronchial nodules which, when enlarging, have reached the mucous membrane, caseated, and

[1] De hérédité parasitaire de la tuberculose humaine, Paris, 1898.
[2] Der primäre Lungenherd bei der Tuberkulose der Kinder, Berlin, 1912.

PLATE III

Large Irregular Cavity with Shaggy Walls in Upper Lobe, which is Covered with a Thick Pleura.

Lower lobe shows conglomerative tubercles and gelatinous degeneration.

Anthracosis *all* over.

PLATE IV

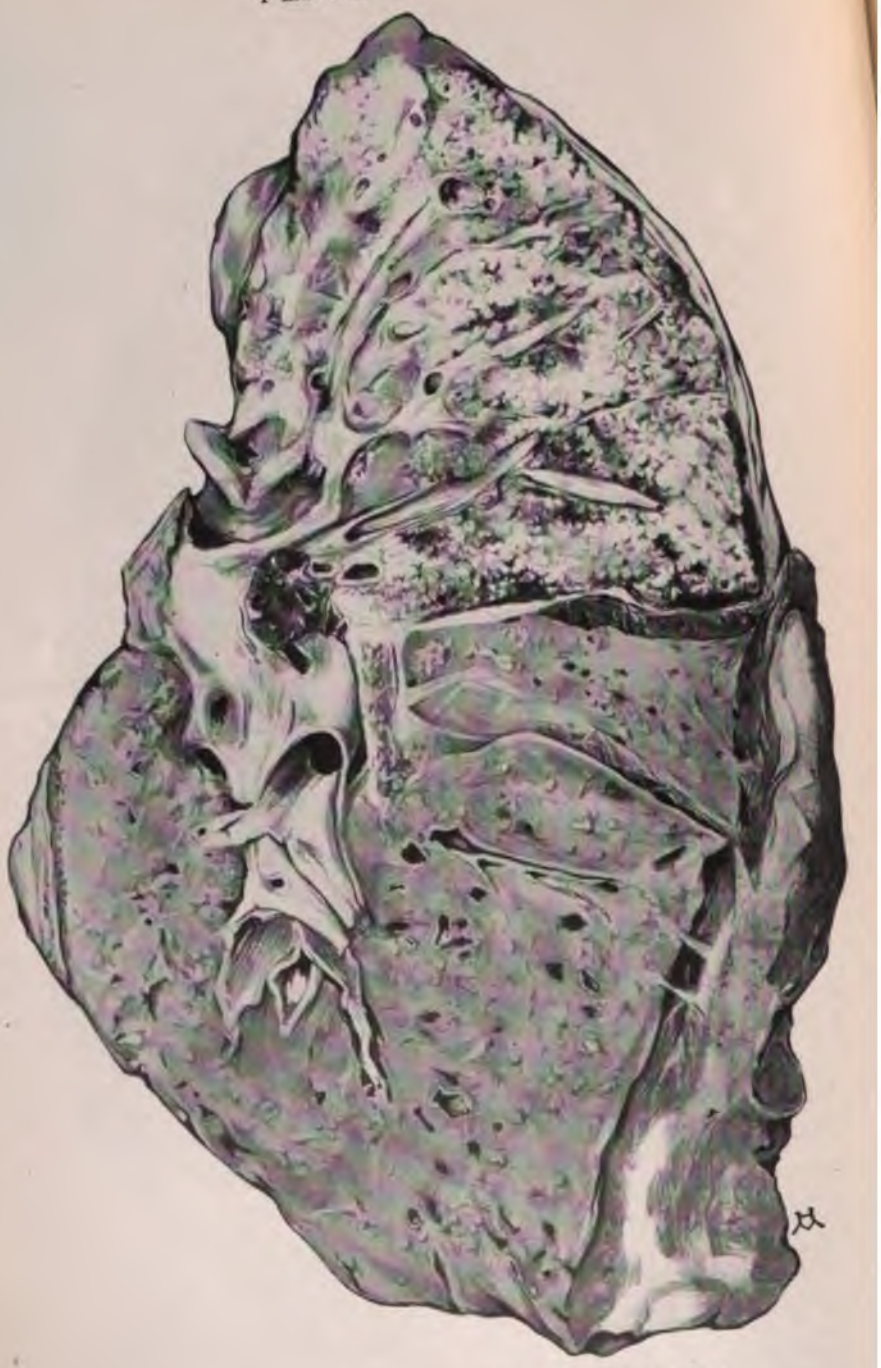
Four Large Communicating Cavities, with Smooth, Glistening Walls and Crossed by Vascular Bridges. Pleura Very Much Thickened.

PLATE V

Enormous Excavation of Nearly the Whole Lung.

The wall is smooth, but traversed by thick bridges. Bronchial glands enlarged and calcified.

PLATE VI



Caseous Pneumonia in Upper Lobe.

Bronchi widely dilated. Miliary tubercles in lower lobe. Enlarged bronch
glands, Pleura thick and covered with miliary tubercles.

produced ulceration. As was already stated in Chapter V, the problem whether phthisis is hematogenous or bronchogenous in origin rotates around this point, to a large extent. The experiments of Bacmeister have shown conclusively that such lesions may be produced by the hematogenous route, and that the primary lesion is not commonly in the mucous membrane. But this does not exclude infection of the mucous membrane. We have already shown that the bacilli may be deposited on the bronchial mucous membrane and pass through the lymph channels into the subepithelial tissue where they take root, without producing a lesion at the point of entry.

Caseous Pneumonia.—The nodular formations are not the only changes wrought by the tubercle bacilli in the lungs. There are also seen larger primary infiltrations which are pneumonic in character; in fact, these distinguish phthisis from pure tuberculosis. These areas are of variable size, from that of a pea to that of an egg, or even larger. They are round, oval, leaf-shaped or lobular in arrangement; they may be single, or several may be found clustered together. They are pale, grayish and, later, muddy in color; at times they look like cheese. They are found in rapidly progressing fibrinous exudations which caseate quickly—caseous pneumonia. Real lobar caseous pneumonia is exceedingly rare. The diseased parts are voluminous, airless, heavy, like in the hepatization of lobar pneumonia.

Microscopically, there is found an albuminous mass in which fibrin, red blood corpuscles and alveolar epithelium may be discovered, but the alveolar structure may still be made out at an early stage. When seen in the early stage we can follow the rapidly ensuing process of coagulation necrosis in the alveolar septæ. Tubercle bacilli are found in large numbers, especially at the periphery of the cheesy focus. The final result is always the breaking down of the caseated and degenerated débris, leaving excavations, which will be discussed later on, excepting when the process involves but a very small area, and some authors say that a cure is then possible by absorption of the caseous matter.

Caseous pneumonia cannot always be differentiated from nodular tuberculous lesions, because when the nodules extend rapidly, as they do in some acute cases, they consist mainly of a conglomerate group of alveoli filled with exudate; the more rapidly the process progresses, the more they are coalescing and the greater the similarity to caseous pneumonia.

Beitzke[1] points out the main differences between tubercle and caseous pneumonia as follows: Caseous pneumonia is an exudative inflammation, while tubercle is a productive one. In the former there are therefore found loose exudate cells and fibrin, while in the latter solid tissue is found, and fibrin is almost never encountered. The exudate in caseous pneumonia lies in the lumen of the alveoli, while the tubercle

[1] In Aschoff's Spez. pathol. Anatomie, Berlin, 1913, **2**, 299.

is located in the interstitial tissues. In caseous pneumonia the elastic fibers remain intact, while the granulation tissue of the tubercle destroys them. These differences show the necessity for differentiation between the two processes. But etiologically they cannot be separated: Both are due to the same cause, both combine and affect the lung tissue, so that only the microscope can decide the intensity with which each is represented in a given lesion.

Localization and Fate of the First Lesion in the Lung.—The first lesion cannot be recognized at autopsies of cases of old chronic tuberculosis, and it cannot be definitely determined whether the disease has arisen by the hematogenous or aërogenous route, as has already been mentioned. It appears, however, that the initial lesion heals in the vast majority of cases. It may also happen that the initial lesion should be completely, or partly, healed in one lung, while the second lung becomes affected with progressive disease. The nodules undergo complete fibrous replacement, become surrounded by connective-tissue which often implicates the surrounding overlying pleura, a cicatrix is formed which contracts the affected part of the lung, resulting in those puckered scars so often seen at autopsies. Inasmuch as the lymph channels are obliterated, the pigment particles inhaled with the inspired air cannot be removed, and they remain in the connective tissue, thus causing slaty induration.

This mode of healing is not the rule. Often, though the focus caseates it is nevertheless surrounded by a fibrous capsule; the caseous center then softens, as has already been described.

Extension of the Lesion.—The morbid focus may erode a blood-vessel and thus break into the circulation, causing acute general miliary tuberculosis, but this is comparatively rare, perhaps because of thrombosis of the supplying vessels. Usually the process extends by the invasion of the tissues in the immediate neighborhood of the initial tubercle. Even when some sclerosis takes place, or the old tubercles calcify, the extension may proceed unabated. Conglomerate tubercles, massive infiltrations which are complicated by pneumonic processes, are thus evolved.

The bacilli spread along the lymph spaces and lymph channels from the areas which have undergone pneumonic changes. This is proved by the fact that around old lesions there is often found a crop of new tubercles. In the same manner occur fresh lesions in the neighborhood of old scars or calcified areas in the apex. Formerly it was thought that the latter are caused by new infections, or superinfections, but since we have learned about the immunity of the tuberculous to new exogenous tuberculous infections, we consider these as metastatic endogenous extensions of the process. These metastatic tubercles increase in number, coalesce, and finally caseate.

At times the extension of the process proceeds along the peribronchial lymph channels and the result is a lobular arrangement of the focus, often looking like a mulberry. Some of these lesions, especially

when exudation takes place, simulate the bronchopneumonic picture very much.

Metastatic extension of the process may also occur along the bronchial tubes and then it runs a rather acute and progressive course. When a necrosed focus reaches the inner surface of a larger bronchus and breaks through the mucous membrane, the caseated matter is carried along the lumen of the tube and may be coughed out. But at times it is aspirated into the alveoli where it may produce a lesion of nodular type or a rapidly caseating pneumonic consolidation. Inasmuch as in such cases we deal with larger numbers of bacilli, they may be distributed over larger areas. Most of these aspiration infections occur in the lower lobes of the lungs, but the metastatic infective matter may be carried to the apex by vigorous cough. These metastatic autoinfections may produce disseminated tuberculosis, but in the majority

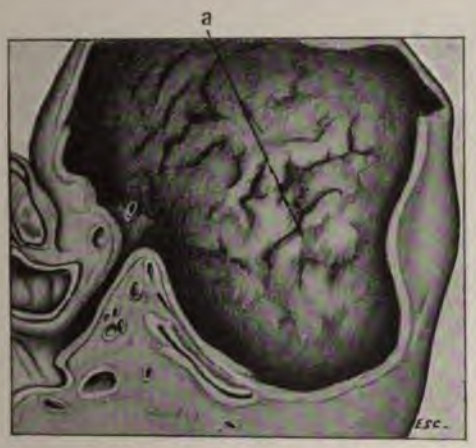


FIG. 26.—Tuberculous cavity (*a*) at apex of lung, showing its relation to a bronchus. (Adami and McCrae.)

of cases a single area is infected and the lesion extends by contiguity, or is of the caseous pneumonic variety; in others indurated nodules result.

Dr. J. Kingston Fowler[1] has given in detail an account of the usual course of the secondary deposits in chronic or subacute phthisis as he found it while making numerous autopsies. He found that the first deposit of tubercles is not at the extreme apex. It is most commonly situated from an inch to an inch and a half below the summit of the lung and rather nearer to the posterior and external borders, and spreads backward, this line of extension explaining the fact that the physical signs of tubercle are often first noticed over the supraspinous fossa. In front, the lesions correspond to the supraclavicular fossa

[1] The Localization of the Lesions of Phthisis, London, 1888.

or to a spot just below the center of the upper lobe, about three-quarters of an inch within its margin, and perhaps separated by an inch or more of healthy tissue. The second and less usual seat of the primary lesion is somewhat lower and more external, and corresponds to the first and second interspaces at the outer third of the clavicle. The lesion extends downward. The part which next shows tuberculous deposit is the apex of the lower lobe (the middle right lobe being passed over), from an inch to an inch and a half below the upper and posterior

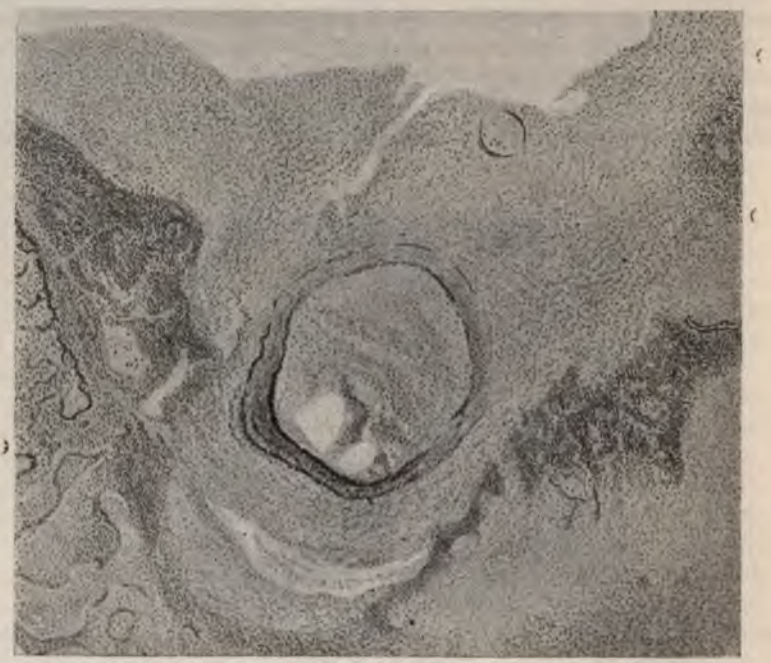
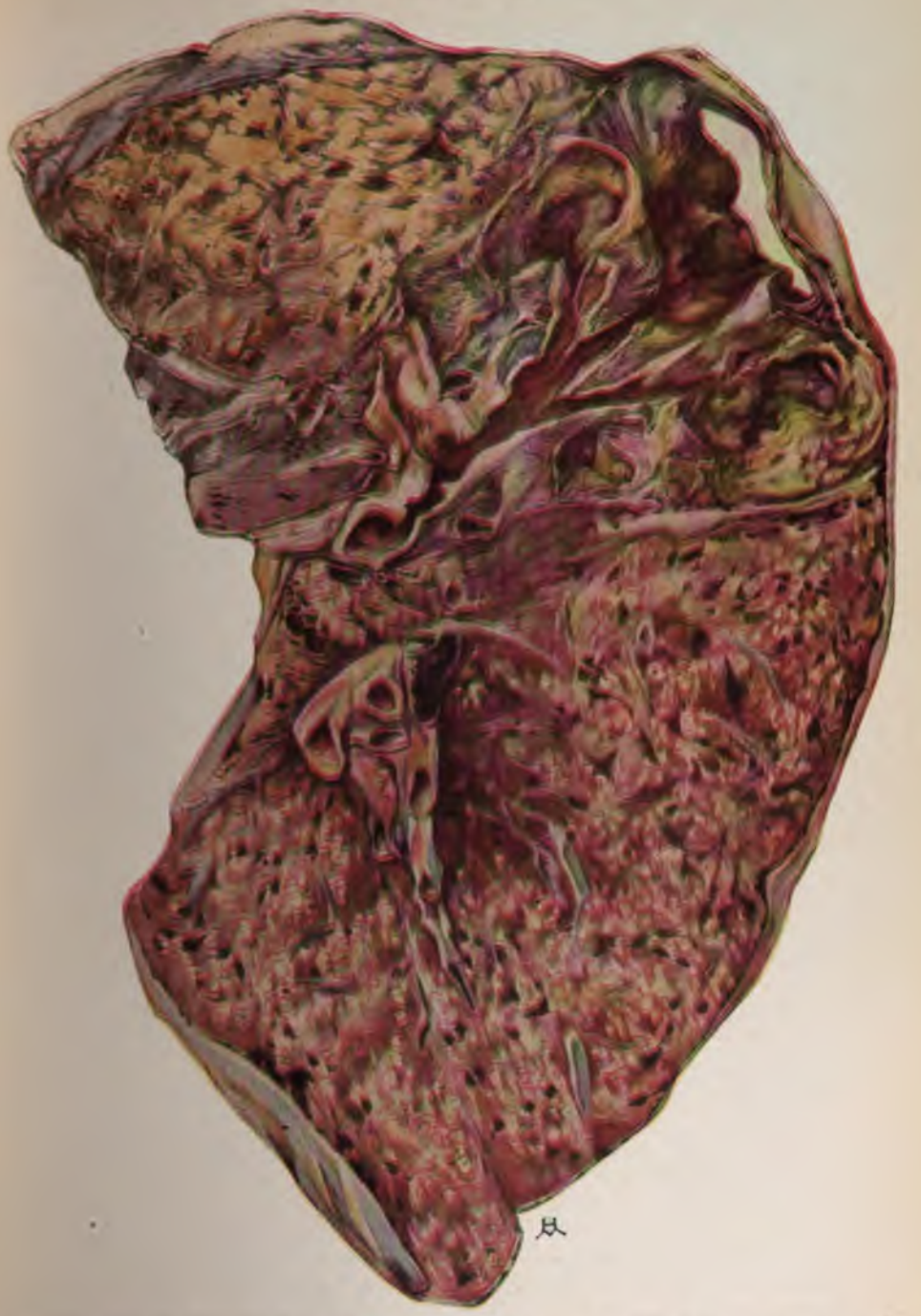


FIG. 27.—Wall of a pulmonary cavity. The upper part of the section shows tissue undergoing caseous degeneration, in which may be noted the following points: leukocytes whose nuclei have, at least in part, retained their staining properties; an obliterated vessel, some of the elastic tissue of which still persists; finally, a pulmonary arteriole almost blocked by endarteritis, the upper part of the vessel being included in the caseous coat of the cavity and in the process of tuberculous necrobiosis. (Letulle and Nattan-Larrier.)

extremity, and about the same distance from the posterior border, a spot nearly corresponding to the chest wall opposite the fifth dorsal spine, midway between the scapular border and the spinous processes. This lesion tends to spread backward toward the posterior border of the lung, and laterally along the interlobar septum. The extension in the lower lobe is almost always from above downward and by islands of deposit of racemose shape with healthy lung between. The second lung is seldom the seat of secondary deposits until the lower lobe of the first lung attacked is implicated. The lesions are usually

PLATE VII



Voluminous Tuberculous Lung with Large Cavity Communicating with Main Bronchus.

Bronchiectasis. Hilus glands enlarged. Lower lobe studded with miliary and softened tubercles.

located in the same situations as those of the apex of the opposite side, but sometimes their site is close to the interlobar septum, midway between its upper and lower extremities, corresponding to the upper axillary fold. Extension in the lower lobe of the second lung follows the course of the lesions in the lower lobe of the first lung.

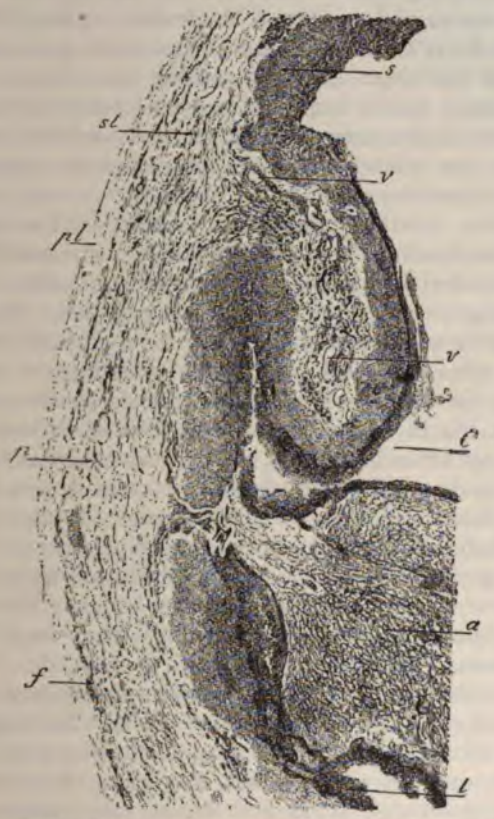


FIG. 28.—Large subpleural pulmonary cavity. *pl*, thickened visceral pleura; *p*, subpleural pulmonary parenchyma transformed into fibrous tissue; *f*, groups of leukocytes accumulated under the visceral pleura; *sl*, fibrous tissue under caseous masses which delimit the wall of the cavity; *s*, caseous masses formed at the expense of the pulmonary parenchyma and representing the zone of extension of the cavitary lesion; *v*, *v*, pulmonary vein placed in the center of projections which partition the cavity (remains of the interlobar framework); *l*, purulent masses loaded with bacilli attached to the surface of the cavity; *a*, pulmonary parenchyma not yet invaded by tuberculous caseation. (Letulle and Nattan-Larrier.)

Cavitation.—When the caseated and softened detritus, affected by certain chemical changes, becomes undermined in various directions, blocks of dense tissue are loosened and cast off, then expectorated, leaving vacant areas in the lungs which communicate with one or more bronchi. The walls may appear sinuous, pouchy and covered with

caseous or purulent material and detritus of disintegrated tissues, or covered with a pyogenic membrane. In some cases they are smooth and glittering, all of which depends on their mode of origin.

The excavations in phthisis may be single or multiple, and they are mostly located in the upper parts of the lungs, the apices. They may be the size of a hemp seed to that of a fist, and in rare cases the complete lung is excavated, leaving a thick shell of the pleura. William Ewart[1] pointed out that excavation is especially prone to attack definite regions of the lungs. The apex of the lower lobe is thus affected at a date anterior to the implication of the lower parts of the upper lobe. The base and anterior border of the lower lobe are least prone to excavation, just as these parts are altogether the last to be involved in the tuberculous process.

The question whether true *bronchiectatic cavities* may occur in phthisis has been debated. Ewart denied such a possibility, and when found, he considers it purely secondary to the undue strain thrown upon the spongy structures which escaped disease. But more recent investigations have shown that they may occur. Delafield and Prudden found them very frequently. The superficial layer of an affected bronchus may be cast off while the process of caseation goes on in the deeper layers. In fact, cavities may be formed without the destruction of the inner bronchial lining. When the tuberculous process proceeds slowly and proliferation of tissue is more active than necrosis, the bronchi dilate cylindrically and, because the more resisting elements—cartilage and elastic fibers—perish, only an unsupported, smooth or slightly ulcerated mucous membrane remains, which yields to the expiratory pressure of the air during cough. These excavations are usually cylindrical or round in shape. They may be considered true bronchiectatic cavities.

When multiple, the separating walls of cavities may be gradually destroyed and a sinuous vomica is thus formed. The large vessels and the affected bronchi resist the destructive process for a long time and remain as cylindrical trabeculæ, traversing the cavity in various directions. These tough septa and bridles are, however, not always remnants of persisting bronchi and bloodvessels. William Ewart has shown that they are more often chiefly composed of condensed airless lung, representing the remains of collapsed alveolar tissue originally separating discrete cavities. When finally these are also destroyed, only ridges and stumps of fibrous tissue remain within the cavity, and also septa which separate accessory excavations communicating with the main cavity.

Only a small proportion of the cavities are bronchiectatic in origin; the vast majority arise through the hepatization and caseation of pulmonary parenchyma and expulsion of the necrotic tissue by expectoration. They have irregular, ragged walls on which there are attached

[1] British Med. Jour., 1882.

pieces of necrotic tissue of various dimensions, bands separating remnants of interlobular septa of the lung. Within the cavity there are often found some large necrotic lumps of tissue, or sequestra, which are too large to be expelled through the communicating bronchus. On rare occasions a cavity is formed when a large part of caseated pulmonary parenchyma is sequestrated *in toto*. In case the cavity is derived from a small caseous peribronchial or bronchopneumonic focus, it is small, more or less circumscribed and round. But when it is derived from a larger pneumonic process it is large from the start and irregularly limited. But small excavations may fuse, coalesce, and form large, pouchy cavities. The septa which separate them fade away and a large, ragged excavation is formed; its walls are covered with a pyogenic membrane, consisting of granulation tissue and secreting tuberculous pus, like a chronic abscess.

William Ewart thus describes the walls of tuberculous cavities which have been freed from secretions and débris: Internally the surface is lined with a grayish false membrane, often of appreciable thickness, but in other cases possessing a little more substance than the bloom of a fresh fruit. In either case it is readily detached and exposes a layer which constitutes the inner and vascular portion of the capsule, the outer portion of which is purely fibrous. The relative thickness of these three coats varies according to the age of the cavities and to the degree of irritation under which they may be placed. The chief features of tuberculous cavities are: (1) Absence of protecting epithelium; (2) gradual decay, leading to the formation of a necrotic layer (pseudomembrane); (3) gradual fibroid growth from without constituting the so-called capsule.

Formerly it was stated that cavitation invariably implies mixed infection. T. Mitchell Prudden's[1] experimental investigations have shown that injections of pure cultures of tubercle bacilli into the trachea of guinea-pigs and rabbits produced pulmonary infiltrations; when streptococci were added, cavitation was produced. But more recent investigations tend to show that tubercle bacilli alone are capable of producing excavations. In this country Ira Ayer[2] found cavities in the lungs of rabbits after injecting intratracheally massive doses of a suspension of tubercle bacilli containing many coarse clumps. Bacmeister's experiments also showed that in animals in which tuberculous infection produces no cavitation, pressure on the apex will produce it, and that mixed infection is not necessary for the purpose. The pyogenic microörganisms found in the walls and secretions of tuberculous cavities are now explained as secondary implantations of these organisms after cavitation has taken place as a result of the action of the tubercle bacilli.

In slowly progressing or stationary cases a wall of connective tissue, even of non-tuberculous granulation tissue, may form around the

[1] New York Med. Jour., 1894, **60**, 1.
[2] Jour. Med. Research, 1914, **25**, 141.

excavation, and the necrotic parts within are cast off and expectorated, leaving a smooth cavity. On the other hand, in progressive cases, the necrotic process digs itself deeper and deeper into the parenchyma and the cavity keeps on enlarging and may attain extensive dimensions. With this process, non-tuberculous infection often takes place through the invasion of streptococci and staphylococci and other microörganisms which invade the walls. Here mixed infection is frequently very effective in extending the diseased area. The pleural layers over superficially located cavities are usually united by dense adhesions.

These cavities have a tendency to enlarge in the manner just described, but on rare occasions they may shrink because of vigorous sclerosis around the lesion which causes contraction. It is more common that the walls should remain smooth and quiescent for many years and, like a chronic abscess, discharge externally through a narrow sinus. But even caseous, ragged cavities may expel the necrotic tissue completely and permit the proliferation of connective tissue around the walls. Healing may thus result, the spongy condition of the adjacent lung favoring contraction. But such a course is less likely to occur when the excavation is extensive, owing to the surrounding caseous pneumonic processes which usually show a tendency to progressive decay.

In extreme cases in which the excavations are extensive and the formation of connective tissue is vigorous, implicating the subpleural structures, the entire lung may be destroyed and reduced to the size of a man's fist. In these cases the diaphragm is pulled upward and with it some of the abdominal viscera, especially the liver and stomach. The mediastinum is pulled over to the affected side, pushed along by the unaffected emphysematous lung. Complete dextrocardia may be found in such cases, with the tuberculous lesion in the right lung; in left-sided lesions the heart is often pulled to the left and upward.

Closed Cavities.—Occasionally cavities are found in the pulmonary parenchyma which do not communicate directly with a bronchus, either because the lumen is occluded with the products of the exudate, or connective tissue has proliferated just at that point and plugged up the passage to the bronchus. Such a closed cavity may open up secondarily when the plug is removed from any cause. Perfectly closed cavities in the anatomical sense are not frequently encountered by pathologists, at any rate not so frequently as clinicians make such a diagnosis.

Aneurysms of Rasmussen.—When the process of caseation and softening involves one of the bloodvessels, which very often traverse the walls of cavities, ulceration may extend to the vessel, causing profuse and fatal hemorrhage. The walls of the exposed vessel become thinner and thinner and finally erode. Because of the loss of support due to the progressive inflammatory decay of the surrounding pulmonary parenchyma, it finally yields to the intra-arterial blood-pressure.

PLATE VIII

Acute Progressive Phthisis.

Patient succumbed to a brisk pulmonary hemorrhage. Lung honeycombed with cavities; very large cavity in upper lobe. Most of the smaller cavities, as well as the communicating bronchus, are filled with clotted blood. Hilus glands enlarged and caseous. Pleura thick and adherent.

More frequently hemorrhage occurs after the formation of an aneurysmal dilation of some branches of the pulmonary artery traversing the walls of the cavity (Fig. 29), first described by Rasmussen. The diseased arterial wall yields to the pressure, gives in first without rupturing owing to the withdrawal of support of the exposed side, and a sacculated aneurysm results; rarely a fusiform aneurysm results from the uniform dilation of the artery. Douglas Powell points out that the fibrotic cavities of old standing are more likely to develop

FIG. 29.—Aneurysm of Rasmussen. The cavity is cut at two points and shows the wall (*c*) with a cavitary projection (*a*), the aneurysm, which is ruptured above. The blood has coagulated in the lower part of the aneurysmal sac. The wall of the cavity has a caseous lining which is continued into the aneurysm. Several pulmonary veins (*p*), included in the caseous lining, have been obliterated and can only be recognized by the remains of the elastic fibers. (Letulle and Nattan-Larrier.)

aneurysm, and that aneurysm is more especially found on the exposed side of vessels which are partly buried in indurated tissue. It is difficult to discern these aneurysms in most cases which come to autopsy because the cavities in which they are located are flooded with blood. Only after thoroughly washing the cavity may they be detected as white, round, sessile projections from the caseous wall of the excavation. They vary in size from that of a pinhead to that of a pea. Exceptionally they are of the size of a plum. They are usually single, but there may be found more than one and, in rare cases, more than

twenty have been found in one lung. In exceptional cases healed aneurysms of Rasmussen have been found in the tuberculous cavities.

Because organized clots and thrombi obliterate the vessel, hemorrhage is comparatively rare unless these aneurysms form. In small cavities the effused blood may by itself prevent further hemorrhage, providing the communicating bronchus is temporarily plugged, or is naturally of a narrow caliber. But many cavities are large and when a vessel ruptures, hemorrhage of great violence takes place.

Rupture of a Cavity into the Pleura.—When a rapidly progressing excavation is located superficially in the lung and reaches the surface the pleura may caseate and rupture. In acute cases in which there is no time for the formation of adhesions between the pleural layers, a loss of continuity in the latter opens up a cavity and permits the escape of its contents, as well as air, into the pleural cavity. Pneumothorax is the result, and when this has lasted for some time, serous and purulent effusions—hydropneumothorax, pyopneumothorax, etc., are formed. These are quite rare in slowly progressing cases of phthisis because adhesive pleurisy results before rupture of an excavation takes place. In old cases I have observed that when pneumothorax does occur the rupture usually takes place into the pleura of the side that was only recently implicated.

Reparative Processes.—We have already spoken of the process of repair that goes on hand-in-hand with the process of destruction in phthisis, and which is found to a certain degree in all cases excepting those of the most acute types. Judging by the large proportion of persons who at the autopsy are found with fibrous scars in the lungs and pleura, as well as with calcified foci in the parenchyma and glands, it becomes a convincing fact that more tuberculous lesions in the lungs are healed than progress to caseation and softening. It has also been found that many cases of these "healed" tubercles contain virulent tubercle bacilli and thus remain a constant source of danger: They may flare up at any time and again begin to progress, or by metastasis create new tuberculous foci in adjacent or distant parts of the lungs or other organs.

Tendeloo[1] gives the following details about the reparative processes in pulmonary tuberculosis:

1. Every fibrous focus is to be considered as an old tuberculous lesion.

2. Calcification removes all danger of the further spread of the lesion. (This is not in agreement with the views expressed above and which are accepted by most authors.)

3. A fibrous capsule separates quite effectively its caesous contents from the rest of the parenchyma of the lung, and the process may remain quiescent for a long time. So long as there remains caseous matter within the capsule, or non-fibrous tuberculous tissue, there is always danger that the caseous focus may extend beyond the fibrous

[1] In Brauer, Schröder, and Blumenfeld's Handbuch der Tuberkulose, 1915, **1**, 98.

capsule, and also that the decay of the latter may favor a further extension of the tuberculous process by growth and metastasis. So long as the bacilli remain virulent in the lesion, and there are connections between the contents of the focus and the surrounding pulmonary parenchyma through lymph spaces, they can grow under certain circumstances and induce pathological changes in other parts of the lung. On the other hand, a fibrous capsule interferes with medication reaching the lesion.

FIG. 30.—Primary caseous focus in the left upper lobe with miliary tubercles in its vicinity. Caseation of the regional lymph nodes of the left upper lobe. Caseation of the upper tracheobronchial lymph nodes. Acute miliary tubercles in the lower tracheobronchial lymph nodes. Over both lungs disseminated tubercles are to be seen. The upper tracheobronchial and bronchopulmonary lymph nodes in the right side are free from pathological changes. (Anton Ghon.)

4. A fibrous capsule has the same significance for an excavation. But in this case other dangers are added: So long as the cavities contain caseous matter, bronchogenous metastasis is threatening because there are always virulent bacilli in the caseous matter. The dangers of softening are greater in excavations communicating with the bronchi because the air has free access to their contents and may bring in other microörganisms, thus causing mixed infections.

5. Healing of a cavity is possible when it is cleared of its contents and the walls granulate. Small vomicæ may heal when their contents are evacuated and the walls shrink. In more extensive excavations there always remains some vacant space. When no open lesion remains, the elastic fibers and bacilli disappear from the sputum.

Ewart points out that whereas in other the organs the obliteration of abnormal spaces is effected by free granulations arising from the bottom of the cavity, surface granulations are practically absent from tuberculous excavations. Still, he holds that, if freely drained, they may granulate successfully and the walls finally adhere. This is in agreement with the more recent views of Tendeloo. But this is more likely to be seen in small vomicæ, while in the large ones the air and fluid contents offer obstacles to perfect contact of the surfaces.

In general, we may consider the productive tissue changes as salutary, while the degenerative—caseation and softening—as phenomena lurking with dangers. Still, even in the latter healing is possible through calcification, or the removal of the products of tissue disintegration from the air passages. It is doubtful whether caseous matter can be absorbed, though some insist that this is possible. Exudative tuberculosis may terminate favorably or unfavoraby, according to its progress along the lines of absorption, or in other forms, caseation and softening, and elimination with the expectoration or by calcification.

It thus appears that even extensive tuberculosis may become quiescent, although we cannot speak of healing and *restitutio ad integrum* in the anatomical sense. It must always be borne in mind in this connection that the anatomical changes are not the only ones which decide the outcome of the disease in most cases.

Emphysema.—The unaffected parts of the lung in chronic phthisis often show emphysematous changes; in fact, occasionally on removing the lungs from the thorax after death, they may be found so voluminous that the tuberculous lesions is not seen without a search. The surface of the emphysematous parts of the lung is usually puckered because of the traction exerted by fibrous bands and excavations within the organ; or, in localized emphysema, which is more frequent, the surface shows bullæ of various sizes.

This emphysema is compensatory. When one lung is extensively involved by the tuberculous process, the other undergoes vicarious enlargement, at times encroaching beyond the middle line; when both lungs are affected, the unaffected parts become emphysematous. It appears that this is strictly for the purpose of enlarging the alveolar surface of the parts which remain intact thus increasing the breathing surface. In fact, microscopic examination of the emphysematous parts of the lung shows that there is no degenerative atrophy of the alveolar septa and bloodvessels, as in true emphysema. The alveoli are simply distended.

PLATE IX

Liver Showing Amyloid and Fatty Degenerative Changes.

The Pleura.—The pleura is implicated in nearly every case of phthisis. A large proportion of cases are preceded by pleurisy, moist or dry, but even then it is usually secondary to extension of some small lesion in the lung. Pleural adhesions are found at the autopsy in nearly all fatal cases of phthisis, excepting those running an exceedingly acute course. In some cases they are so dense and compact that it is difficult or impossible to remove the lungs without injuring the pleura. Sometimes the pleura is thickened all over; in many only partly, especially over the seat of the main lesions, and also at the base where thickening of the diaphragmatic pleura is not uncommon with resulting elevation of the diaphragm. Many fibrous bands are often seen extending from the pleura into the parenchyma of the lung. The adhesions may be lax and easily separated, but in many cases they are dense, and when extensive the thick pleura may surround the lung like a shell. In rare instances the pleura is even found calcified in places, or very extensively. Very frequently thickening of the pleura between the lobes of the lung is found. All these adhesions are great hindrances to the induction of artificial pneumothorax for therapeutic purposes. On the other hand, they prevent the occurrence of spontaneous pneumothorax through rupture of the visceral pleura over the site of superficially located pulmonary lesions, and when pneumothorax does occur, it is only localized. Serofibrinous pleurisy is quite frequent and, in fatal cases, exudation occurs in a large proportion shortly before death.

The Larynx.—Primary tuberculosis of the larynx is exceedingly rare, but this organ is sooner or later affected in about one-third of all phthisical patients. While some of the lesions may occur as a result of inoculation by the sputum, it seems that the most common mode of access of the bacilli from pulmonary lesions is *via* the lymphatics. This path of infection is plausible anatomically, and is supported by the fact that the earliest and predominating lesions are usually on the side of the larynx corresponding to the more extensive and active lesion in the lung.

The process starts by the formation of multiple miliary nodules directly under the mucous membrane. These coalesce and undergo the usual fate of caseation and breaking down, with loss of substance and formation of irregularly outlined ulcers. Should the perichondrium be reached, and it is in a large proportion of cases, necrosis of the cartilage results. While any part may be involved, the epiglottis, aryepiglottic folds, and true and false vocal cords are sites of predilection. Occasionally, shallow ulcerations of the trachea result from inoculation by the sputum.

The Intestines.—The intestines are only rarely the seat of primary tuberculosis. In children it has been found in between 30 and 50 per cent, and in adults Orth and Henke found it in 3 to 5 per cent of all autopsies. But in phthisis they are secondarily affected to the extent of 90 per cent of cases, according to some authors. Some of the anatomical changes are merely tuberculous nodules, but in most

there are found ulcerations of the mucous membrane of the ileum and to a less extent of the jejunum and ascending colon (Fig. 30). These ulcers heal but rarely, though occasionally there is encountered a case of stricture of the intestine due to a contracted scar resulting from a tuberculous ulcer. On the other hand, these ulcers may perforate into the peritoneal cavity with the usual results of these accidents. Ischiorectal abscesses are very frequent in phthisical patients.

These ulcerations are not so closely restricted to the lymphoid nodules as those of typhoid, and they also differ from the latter by having their long diameter extending transversely to the direction of the gut, so that they are often known as girdle ulcers. Before the intestine is opened the location of such an ulcer can be surmised by the linear arrangement of tubercles along the dilated lymphatics which are plainly visible through the serosa. The depth of the ulcers varies; they may extend down to the submucosa, muscularis or serosa, or they may perforate with the usual results of these accidents. Another form

FIG. 31.—Tuberculous ulcerations of the intestines. (Tendeloo.)

of intestinal tuberculosis—the so-called hyperplastic form—results in the formation of a mass of scar tissue in the neighborhood of the ileocecal valve, so that a large tumor is palpable, and symptoms of obstruction result.

The Peritoneum.—Pulmonary tuberculosis is also quite frequently complicated by tuberculous peritonitis. The bacilli may reach the peritoneum *via* the blood stream, or they may be deposited from a perforated intestinal ulcer, or a caseous mesenteric lymph gland, or a Fallopian tube. Depending on the number and mode of entrance of the bacilli, as well as on the resistance of the individual, the peritonitis may be localized or generalized. It may be almost purely plastic with the formation of dense adhesions, or there may be a large ascitic effusion. In some cases there are localized purulent effusions walled in by dense adhesions. Some authors believe that the localized thickenings of the capsules of the spleen and liver sometimes observed at autopsies are the results of a former tuberculous peritonitis.

Pathological Changes in other Organs.—It is exceedingly rare, if it ever occurs, for a case of pulmonary tuberculosis that leads to a fatal termination to run its course without implication of organs other than the lungs, pleura and larynx. Of course, all organs of the body will undergo, to a greater or lesser degree, the non-specific metabolic changes incidental to any long-standing toxemia, such as atrophy, fatty degeneration, etc. Moreover, the amyloid change that is prone in certain of the viscera during the course of chronic suppurations, is most frequently found in victims of tuberculosis, particularly of the bones and joints.

But more important, perhaps, than any of the above-mentioned generalized, non-specific, nutritive disturbances are the specific lesions resulting from metastasis of tubercle bacilli to other organs of the body

FIG. 32.—Miliary tubercles of the serous membrane of the mesentery of the intestine. (Ribbert.)

The bacilli may reach other organs from the lungs by the following routes:

1. By direct extension of the pulmonary lesion, this being the usual mode of involvement of the pleura, as has already been shown.

2. By transport of the bacilli with the sputum. Swallowed sputum is at times, though rarely, the cause of intestinal lesions. The common mode is, however, hematogenous and lymphogenous.

3. By the lymphatics, this being the route of infection of the lymph glands draining the lungs. From the regional lymph glands of the lungs and intestines the bacilli may be very widely distributed *via* the lymph and blood streams. As has already been stated, the laryngeal lesions appear to result from lymphogenous transmission in most cases.

4. Hematogenous transmission. The presence of tubercle bacilli can very often be demonstrated in the blood of sufferers from pul-

monary tuberculosis (see p. 284), and it is not surprising that hematogenous metastasis in other organs occurs, particularly late in the disease. This appears to be the explanation of most cases of genito-urinary tuberculosis, tuberculous meningitis, and other distant visceral lesions.

In this manner any organ of the body can be infected metastatically in the course of pulmonary tuberculosis, but some are much more frequently affected than others.

The glands, especially those in the thorax—the bronchial, tracheal and mediastinal—and of the mesentery, are very often affected in children and adults who suffer from phthisis, more often than is generally appreciated. In fact, it may be stated that the tracheobronchial glands are affected in nearly every case of phthisis. On careful and painstaking search small, microscopic tuberculous foci are often found in apparently unaffected glands; but the majority are swollen, enlarged and many are softened while others are calcified. In children these tuberculous glands very often give no clinical indication of their implication; in fact, it is at times difficult to discover any changes in the bronchi and parenchyma on cursory examination at the autopsy. Still, these glands are frequently a source of trouble, not only in causing symptoms of tracheobronchial adenopathy, but also because these conditions are to be considered the forerunners of phthisis in the adult, though some look upon them as possible immunizing agents against reinfection in later life.

By pressure these enlarged glands may cause stenosis of the main bronchus in children, while in adults it is less likely to occur because the bronchi are firmer. But the smaller bronchi may be compressed in adults as well as in children. In the latter, suppurating glands at times perforate the trachea, bronchi, pericardium, or esophagus, causing sudden death, tuberculous bronchopneumonia, etc.

The mesenteric glands are only rarely affected in adults, even in those who have tuberculous ulcerations of the intestines, but in children they are often found to be the seat of tuberculous changes, particularly with bacilli of the bovine type. This is in agreement with certain facts discussed in Chapter V. In primary infections the regional glands are invariably implicated. In secondary, or metastatic, infections the glands remain unaffected, as a rule. This rule holds good for the thoracic as well as for the abdominal glands.

The Genito-urinary Organs.—The genito-urinary organs are quite frequently involved. Infection of the genito-urinary system secondary to pulmonary tuberculosis almost always takes its starting-point in either the kidney, or in the epididymis, or the Fallopian tubes, the other organs of reproduction and urination becoming implicated from these by extension or contiguity. These appear to be points of least resistance when hematogenous tuberculous infection is considered.

Bones and Joints.—It is very uncommon that the bones or joints should become involved during the course of pulmonary tuberculosis, though in rare instances it is observed. The reverse is also true to

some extent: sufferers from osseous and articular tuberculosis rarely have active and progressive pulmonary lesions, excepting as a terminal phenomenon, when acute miliary tuberculosis, in which the lungs bear the brunt of the infection, may occur.

The Muscles.—In fatal cases of tuberculosis the muscles are found pale or brown, atrophied and poor in fat; they appear to participate in the process of emaciation to an extreme degree. Microscopic examination shows brown atrophy, fatty, and other degenerative changes. It appears that the diminution in the volume of the muscles is due to an atrophy of each individual muscle fiber, and not to diminution in their number. Tuberculous lesions are very rare in voluntary muscles, they being probably the part of the body most resistant to tuberculous changes.

Nervous System.—While in rare instances the central nervous system proper contains tuberculous lesions, as large solitary tubercles of the brain, the most vulnerable part is the *meninges*, tuberculous meningitis being not an uncommon termination of pulmonary tuberculosis.

Circulatory System.—The circulatory system does not remain unaffected. The heart is small, weak, and atrophic, the individual muscle fibers being atrophied and fatty. Hypertrophy of the right heart may be seen in cases with extensive shrinkage of the lung with pleural and pleuropericardial adhesions. Verrucose endocarditis is seen in rare instances, but it is questionable whether this is due to the tubercle bacilli themselves, or to their toxins. The consensus of opinion is in favor of streptococci being the cause. In acute miliary tuberculosis miliary tubercles may be found in the myocardium, while in chronic phthisis they are rare. There have been reported some few cases of solitary tubercle of the myocardium. Pericarditis, often with copious effusion, is not uncommon. The bloodvessels also may suffer as a result of the infection. Tubercles in the walls of the arteries, veins or lymph vessels are not uncommonly found, and when they perforate the intima, they set up miliary tuberculosis. The pulmonary veins are the most frequent site of such tubercles.

Amyloid Changes.—In those who have suffered from long-standing suppuration it is common to find deposited in the various organs a peculiar substance which Virchow termed amyloid from its supposed resemblance to starch and cellulose. Amyloidosis is particularly common in tuberculosis of bones, though it is quite often seen in sufferers from pulmonary tuberculosis without osseous implication.

While almost any organ may be involved, those most frequently affected are the spleen, liver, kidneys, and intestines. The spleen may be diffusely affected or, what is more common, the amyloid may be restricted to the Malpighian bodies (sago spleen). An affected liver may present no gross changes if the amyloid is slight in amount, or it may be very much enlarged and firm. The amyloid kidney is large and firm, the amyloid material being principally deposited within the

glomerulus. The adrenals, lymph glands, intestines, etc., may also be involved.

Amyloid possesses the property of staining a rich brown with a solution of iodine and potassium iodide. If strong sulphuric acid is used in addition to the iodine, the amyloid stains dark blue. With methyl violet, amyloid stains violet. Examination of histological preparations shows that amyloid is never deposited in the cells, but always between them, being found in the walls of the small bloodvessels or just outside the endothelium of the capillaries. As it is deposited it is effective in causing pressure atrophy of the surrounding tissues; it also cuts off the blood supply by obliterating the capillaries. The chemical nature and origin of amyloid have been disputed. It has been shown to contain chondroitin-sulphuric acid, and seems to be a combination of this substance with a protein fraction.

In addition to the fatty changes of the heart, which have already been mentioned, the liver is frequently thus affected in pulmonary tuberculosis. The liver and spleen also often show frank tuberculous changes. R. G. Torry[1] has reported 131 autopsies on tuberculous subjects at the Phipps Institute as regards macroscopic and microscopic changes in the liver, spleen, kidneys, etc. In the vast majority of cases that succumbed to pulmonary tuberculosis, tuberculous changes were found in the above-mentioned visceral organs. O. Klotz[2] encountered tuberculous changes in the spleen, in 172 out of 404 necropsies. A large proportion showed that the tuberculous lesions had healed.

[1] Am. Jour. Med. Sci., 1916, **151**, 549.
[2] Ibid., 1917, **153**, 786.

CHAPTER VII.

SYMPTOMATOLOGY OF PHTHISIS—HISTORY OF THE PATIENT.

We have seen that infection with tubercle bacilli does not invariably result in tuberculous disease. *Phthisis implies a preëxisting infection, but the latter may take place without any subsequent clinical manifestation of disease.* The diagnosis of tuberculous infection is a simple matter. The application of the cutaneous tuberculin test tells the story promptly, easily, and unequivocally. The chances of error are insignificant and may be disregarded.

But a positive tuberculin reaction, found in over 90 per cent of humanity, as we have seen above, is by no means proof that the individual suffers from any disease, or needs general or special treatment. It only shows that the individual has been infected with tubercle bacilli at some period of his or her life. The infection may not have done any harm. In fact, we have seen that, in all probability, it has immunized him against a new massive infection, which is difficult to avoid, and which might have produced acute and progressive disease, had it taken root on virgin soil.

What we aim at in our practice is discovering not only tuberculous infection, but tuberculous disease. At any rate this is what the patient wants to find out: Whether he suffers as a result of the infection with tubercle bacilli, and whether any treatment is necessary to save or prolong his life. This information can only be given after a careful and painstaking inquiry into the patient's history, the symptoms he suffers from, and the physical signs elicited by an examination of his chest and other parts of his body, and applying some or all the clinical diagnostic methods which have been the achievement of medicine during the past couple of generations.

Hazards of Hasty Diagnosis.—Realizing that the patient's chances of recovery are greatest when the disease is recognized and treated at its very incipiency, there has been a strong tendency during recent years to treat every "suspect" as one who is actively tuberculous until time and observation prove the contrary. This advice has been given by many writers on the subject and followed by numerous physicians. As a result many innocent persons have been banished to sanatoriums, or to distant climatic resorts; many children have been deprived of an education, many workmen induced to leave their employment, many men of affairs to neglect their business. To be sure, some of these non-tuberculous individuals—"suspects"—have been fatigued and

debilitated and needed a rest, and the error in diagnosis has rather benefited them. But with others things have been different. Many a person known to the writer has been trying to remove the stigma of tuberculosis, which he or she never had, without avail; and tuberculosis is a stigma at present, despite our teachings that a patient who has common-sense and decency is as good, and as harmless, as any other person.

We often meet with people who have spent some months in sanatoriums—from all indications they could have gotten along very well without it—and ever since they live in constant dread lest it will be found out that they had been "consumptives." The stigma of sanatorium residence may later interfere with obtaining life insurance, with contracting a desirable marriage; at times even with obtaining lodging by those who have no blood relatives. In fact it may break up a family. I have known persons who have lost their jobs because some patient who knew them in an institution "gave them away."

A hasty diagnosis among the poor and moderately well-to-do—from which classes the bulk of phthisical patients are being recruited—works even more havoc at times. The results of the maxim, "Treat everyone as tuberculous until *he* proves to you that he is not," can be seen in a city like New York where numerous individuals attend tuberculosis clinics for months, even for years, or go from one institution to another for years, though they fail to present any reliable symptoms of active phthisis. I witnessed the autopsy on the body of a woman who remained twenty-six years continuously in an institution; about one-half the time in a sanatorium, the other half in a hospital for advanced consumptives, where she finally died from pneumonia. Careful examination of the viscera failed to disclose an active tuberculous lesion. I calculated that the community spent, or wasted, over ten thousand dollars on this woman, not including the loss owing to her idleness. We may further mention that during the twenty-six years she kept out of the institution at least forty patients with active disease who might have benefited by the treatment.

Many communities keep on spending considerable sums of money on the maintenance of patients who could be cared for in their homes at a lesser cost, or keep them from work merely because of a suspicion that they are tuberculous. Others break up their homes, commit their children to asylums because of a hasty diagnosis of incipient tuberculosis based on some indefinite symptoms and physical signs. It was found in Germany, France, and England that some patients, passed for admission to sanatoriums because of incipient tuberculosis, were fit for active military service during the war. A large number of ex-sanatorium patients have been admitted to the United States Army and they made excellent soldiers. Fifty per cent of patients in one of our largest municipal sanatoriums have negative sputum; that this is an indication that many are non-tuberculous will be agreed to by everyone who has any experience with tuberculosis. With the anti-

formin method of sputum examination at most 10 per cent of active cases are found not expectorating bacilli.

There appears to manifest itself a reaction against the eager chase for "incipient" cases which may swell the favorable statistics of sanatoriums. Authoritative writers now state emphatically that indefinite physical signs should not be relied on, and urge that only constitutional symptoms of toxemia be taken as criteria for active disease. Edward O. Otis[1] questions the wisdom of relying on "the presence of certain physical signs, definite or indefinite, with no symptoms of bacterial toxemia which are interpreted to mean active tuberculosis, and the patient exhibiting such signs is accordingly removed from his family and employment and consigned to a sanatorium, where there is at least some risk that he may receive a new and active infection. Whereas the individual was in no way ill, and probably never would have developed active clinical tuberculosis."

The harm done to the community by the principle of treating all "suspects" as tuberculous has been shown drastically during the World War. At first physicians examining soldiers thought that they were dealing with their civil patients and were hasty in making diagnoses of tuberculosis. In civil practice these would be admitted into sanatoriums where they would remain for a variable time, and be discharged as cured. But in the army, they were taken to hospitals for observation and the result was that in France of 1000 such men, only 1.5 per cent were found to be actually sick with tuberculous disease, according to Kindberg and Delherm.[2] About 113 of the men were merely troubled with chronic nose and throat conditions. Major Rist[3] stated that out of 1000 men in the French army sent back to a base hospital as suffering with pulmonary tuberculosis, 807 were found to be non-tuberculous. I have had under my care many who have either been rejected by the draft boards, or by disability boards, because of alleged tuberculosis which did not exist. The loss to the army in men and in money due to such hasty diagnoses cannot be overestimated. "The evils of such faulty diagnosis are world wide," says Colonel G. E. Bushnell;[4] "they have been encountered in the armies of Germany and of Great Britain, as well as in that of France. There is the same blame for us if we err on the side of a too minute and pedantic regard for slight changes in breath sounds, or in percussion, for all the world is committing, or until recently has committed, the same mistake, and the standpoint is maintained by so many writers of repute that the unwary are scarcely to blame if they believe that it represents the standpoint of the truth." It is the opinion of Colonel Bushnell[5] that "medical officers should be held strictly responsible for the exercise of enlightened judgment as to causes which may or may not be evacuated from their hospitals."

[1] New Orleans Med. and Surg. Jour., 1914, **67**, 311.
[2] Presse médicale, 1917, **25**, 645.
[3] Jour. Am. Med. Assn., 1917, **69**, 1265.
[4] Medical Record, 1918, **92**, 4.
[5] The Military Surgeon, April, 1918, **42**, 383.

A hasty diagnosis is as dangerous as neglect to recognize active and progressive disease. Delay does not mean sure death of the patient; if he is kept under careful observation, we cannot be too late making a positive diagnosis. The acute and progressive cases will manifest themselves very soon, and delay does not count because treatment in these cases is, as a rule, not very effective. In the slow, sluggish cases the delay of a few weeks hardly ever makes any difference in the ultimate outcome. But pronouncing a patient phthisical when, in fact, he has no symptoms of active disease, is often followed by disastrous results to the patient as well as to those depending on him, and to the community which is charged with caring for its tuberculous dependents. It may be said without fear of meeting contradiction from competent sources that *an incipient case in the full sense of the word does not always mean a curable case, or even a favorable case.* Many cases justly classed as incipient have a worse prognosis than those considered "far advanced" in the conventional classification of the disease.

Elementary Principles in the Diagnosis of Active Phthisis.—Active tuberculosis, or phthisis, manifests itself invariably by symptoms of bacterial intoxication. *If there are no symptoms of constitutional toxemia, the patient may have been infected with tubercle bacilli—and who has not been?—but he is not sick with a disease which needs special treatment, costly to the community, and often ruinous to the patient and his family.* Nor must the patient be isolated from his family, and hospitalized to prevent the dissemination of a disease which he does not have. This is a point which must always be borne in mind before a patient is told that he suffers from incipient phthisis.

There is hardly a conscientious physician who is not skilled in making a diagnosis of incipient phthisis from the constitutional symptoms, even though he may have to leave the localization of the lesion to some virtuoso in physical diagnosis. *There is no active phthisis without fever, cough, tachycardia, languor, night-sweats, hemoptysis, etc. Some or all of these symptoms are found soon after the patient becomes actively phthisical.*

If these elementary points were borne in mind by physicians, the number of mistakes of omission and commission would be reduced to a minimum. In fact, if the propaganda made so assiduously, aggressively and, within certain limits, justly, that to be cured, tuberculosis must be discovered in its incipiency, would have insisted emphatically on the symptomatology of the disease, which can be inquired into, observed, and properly interpreted by every practising physician, all cases coming under the observation of physicians would be detected in proper time. It is wrong to blame the general practitioner for the large proportion of cases which are diagnosed rather late, after he has been taught that certain indefinite physical signs may mean phthisis, and just as often may mean nothing. In fact, the general practitioner may retort by saying that the large proportion of non-tuberculous cases admitted and kept in sanatoriums, as well as the large number of patients "cured" within two or three months in the insti-

tutions, prove conclusively that the specialists are no less fallible in this regard.

Natural Method of Arriving at a Diagnosis.—While in the practice of medicine we must often resort to the deductive method of reasoning when attempting to unravel an obscure case, yet in our attempts at ascertaining the presence or absence of active phthisis, we are on safer ground when applying the inductive method. We must first ascertain the individual symptoms and credit each with its true merit. In other words, all the morbid phenomena must be accurately observed; all the material facts are to be carefully inquired into; and, what is of most importance, the interpretation of the collected facts must be correct and in agreement as regards their relation one to another, and to the probable causes which may underlie the process.

To do this rationally, we must carefully observe the appearance of the patient, go into details about the symptoms which urged him to seek medical advice and also inquire into such subjective symptoms as the average patient is not likely to note unless his attention is drawn to them. When all these data have been carefully gathered and properly evaluated, a physical examination is made to ascertain the objective signs of the disease, and these are correlated with the constitutional condition of the patient, with a view of ascertaining whether he is endowed with sufficient resistance to counteract the ravages of the disease.

History of the Patient.—This is to be minutely inquired into. We find out the condition of health or the cause of death of the patient's parents and grandparents, if he is in possession of the facts, or capable of giving them to us reliably, which unfortunately is only rarely the case. Of particular importance is whether either of the parents was actively tuberculous when the patient was an infant. In case the parents have become actively tuberculous when the patient had already passed childhood, his chances of becoming phthisical are not greater than of those who do not have such an hereditary taint. In fact, there appears to be some evidence tending to show that, contrary to the general opinion, tuberculosis, if it occurs at all in such individuals, is apt to run a milder course than in those who have no family history of tuberculosis.

We should not be greatly influenced by the age of the patient. No age is immune to the disease, but each age period appears to have its own form of the disease: In infants, hematogenous, general tuberculosis is the rule; in children tuberculosis of the glands, especially the tracheobronchial group, the bones and joints; in adults chronic pulmonary tuberculosis; in persons over forty fibroid phthisis, and in aged individuals a very chronic form with a symptomatology and course peculiarly its own, etc.

The occupation of the patient has great influence on the chances of developing active phthisis, as was already shown elsewhere (see p. 129), and should be considered when taking the history. A history of an injury to the chest, especially if followed by hemoptysis, is important.

Preëxisting diseases are to be ascertained in detail. In infants and children active disease is apt to follow in the wake of one of the endemic contagious diseases; in adults, typhoid, influenza, pleurisy, pneumonia, diabetes, syphilis, etc., are of etiological moment; but their importance has often been overestimated by physicians. A history of scrofula during childhood has very little bearing upon active phthisis in the adult, excepting perhaps that if the disease does occur, it is likely to pursue a mild, and exceedingly chronic course. The same is true to a certain extent of previous tuberculous disease of the bones and joints. One has to consider the rarity of old scars on the neck or over joints of phthisical patients; or of active and progressive phthisis in those who have had Pott's or hip-joint disease during childhood. (See Chapter XXX.)

In women the menstrual history is to be gone into, and special attention paid to amenorrhea. It is also to be borne in mind that active symptoms very often appear immediately after childbirth. Here again it is to be emphasized that most women who suffer from active phthisis after gestation have had symptoms of this disease before conception and pregnancy.

A history of exposure to infection should not be overestimated in adults, as has been advised by many writers. We have seen that those most exposed to infection with tubercle bacilli, as the hospital staffs of institutions for the care of tuberculous patients—doctors, nurses, and orderlies—are not more liable to become phthisical than those in other walks of life who do not come into intimate contact with consumptives; nor do the unaffected consorts of tuberculous patients suffer from this disease more than others. It is therefore absurd to expect that a tuberculous fellow-workman is more likely to transmit the disease than a hospital patient to a doctor, nurse or the tuberculous to the unaffected consort. *In my own practice I do not at all give exposure to infection any weight in the diagnosis of active phthisis in adults.* It is different with children, especially with infants. Infants of tuberculous parentage, or who have otherwise been exposed to infection, are very likely to have contracted the disease in an active form. With children over three we should ascertain whether the parent has become actively tuberculous while the child was less than one year old, because if the child was older than three years when the parent began to expectorate bacilli, the chances of primary massive infection of the child are remote.

It is a curious fact that, in attempting to trace the source of infection in children, we often find it is one of the grandparents, suffering from senile phthisis, who is responsible, though he or she is ignorant of the true nature of the ailment, having been told that it is bronchitis, emphysema, asthma, etc.

History of the Present Illness.—Of immense importance is the history of the mode of onset of the present ailment, as well as certain symptoms from which the patient has suffered during his lifetime. Previous attacks of "grippe," "colds," bronchitis, etc., may mean

previous attacks of abortive phthisis and should be carefully considered. On the other hand, if we find that these "colds" are due to hypertrophied tonsils, adenoids, etc., in other words, that they are manifestations of the lymphatic diathesis, pulmonary tuberculosis in an active and progressive form is unlikely. Even if there is a distinct tuberculous lung lesion, the prognosis is very good. Previous attacks of typhoid fever, pneumonia, and particularly pleurisy, etc., may have been mild or severe attacks of unrecognized tuberculosis which have subsided. Having been treated for months for neurasthenia, gastritis, chlorosis, or even malaria, is not uncommonly ascertained in the history.

We should inquire into the symptoms which ushered in the present ailment, with special reference to cough, expectoration, lassitude, languor, particularly in the afternoon, loss of weight, hemoptysis, pleuritic pains, or pleurisy with or without effusion, etc. Of most importance in ascertaining the presence or absence of active disease is fever with its concomitant symptoms—chills, backache, anorexia, tachycardia, etc. Night-sweats are to be inquired into and it should be ascertained whether they occur immediately upon going to bed, or wake the patient at some time during the night. The appetite of the patient is to be ascertained, and whether any loss in this direction has been concomitant with the appearance of other symptoms. If the patient knows, he should tell the fluctuations in his weight for the past several years. The condition of the bowels, especially the presence of diarrhea, is to be ascertained.

Of course, if any sputum is available it should be examined microscopically for tubercle bacilli and elastic tissue. The urine should be analyzed for the presence or absence of albumin, sugar, and casts.

After all these data have been ascertained, we proceed with the physical examination of the patient, and this includes not only a careful examination of the chest by inspection, palpation, percussion, and auscultation, but also all other parts of the body from the top of the head to the toes. We may thus find symptoms and signs confirming the diagnosis of phthisis, or proving that the symptoms of which the patient complains are due to some other cause. The stigmata of phthisis are often scattered all over the body, as will be shown later on.

Above all, it must never be lost sight of that, while there is no active phthisis without constitutional symptoms, *there is no single symptom or sign pathognomonic of the disease*, excepting the expectoration of sputum containing tubercle bacilli, and even this is occasionally apt to mislead. It is *only the combination and correlation of various symptoms and signs which clinch the diagnosis*, especially in obscure cases with negative sputum. This fact by no means interferes with the early recognition of active phthisis, and mistakes are more often due to carelessness in observation than to any other factor.

Importance of the Symptomatology of Phthisis.—In the succeeding chapters the physical diagnosis of phthisis in its various forms will be given its proper place, because only with the aid of inspection, percussion, and auscultation can we localize the lesion and gain important hints as to prognosis and the treatment indicated. The symptomatology of the disease, which has been given a subordinate place in some recent treatises on the subject, will be discussed in detail. The reasons are obvious: The general symptomatology of active phthisis can be ascertained by every practising physician, and its bearings on the presence or absence of active phthisis, especially in doubtful cases, are of more significance than indefinite physical signs. *There may be active phthisis without physical signs revealing themselves even to the best-trained specialist, and even roentgenography may fail to disclose the site of the lesion, while many signs of apical involvement are found in healthy persons. But there is no active phthisis without constitutional symptoms.* This is an axiom which cannot be repeated too often. The symptomatology of phthisis, when properly studied and interpreted, gives information as to the onset of the disease, its activity, tendency, and ultimate outlook. It can be ascertained by any medical man. Inasmuch as it often precedes the appearance of definite physical signs, or the signs elicited with the aid of roentgenography, the symptomatology of the disease is to be ascertained first.

We shall therefore begin with a discussion of the most prominent, and more or less constant, symptoms of active phthisis—cough, expectoration, fever, night-sweats, hemoptysis, anorexia, emaciation, tachycardia, etc. Each of these symptoms will be discussed from the standpoint of diagnosis, differential diagnosis, and prognosis. It is only by a proper appreciation of these symptoms that a diagnosis of active phthisis can be made at any stage of the disease, but especially in the so-called incipient stage; while a prognosis based only on findings during a physical examination and roentgenography is bound to prove disastrous to any practitioner in many cases.

CHAPTER VIII.

COUGH AND EXPECTORATION.

COUGH.

Frequency of Cough.—While cough is the symptom which first attracts the attention of the average patient to his troubles, there has been a question whether there are cases of phthisis without cough. Pidoux stated that cough is the first and last symptom of phthisis; when it is absent, its negative significance is almost absolute. According to many writers, a patient who does not cough is not tuberculous, while there are others who consider it the most constant of symptoms of early phthisis. However, Louis, Wilson Fox, Möller, and others, speak of patients who passed through the disease without ever coughing.

This disagreement is due to various causes. The statement made by many phthisical patients to the effect that they do not cough is to be taken with considerable reservation. Mild cough, clearing the throat in the morning, or hawking, which causes but little annoyance to individuals who are not given to introspection, may be overlooked. Even in the advanced stages, when considerable sputum is brought up, the patient may be under the impression that he does not cough—the sputum is carried by the cilia of the bronchial and tracheal mucous membranes, and when it reaches the vocal cords it is easily removed without effort, or swallowed. In the latter case the patient may not even expectorate. I have seen this to be the case with many patients, especially females. For this reason, it is often ascertained by close questioning that there is a little, mild cough, "just like everybody else coughs." I have, however, seen many patients in whom physical exploration of the chest was negative for quite some time, but the continuous cough, productive or unproductive, was the only symptom which urged them to seek a diagnosis, and excited a careful study of the case by the physician.

Another class of patients who do not cough despite active tuberculosis are aged persons, of whom details will be given later on. The same is true of some cases of phthisis with cavities—mouthfuls of sputum may be brought up without any effort, or cough, as in bronchiectasis.

Cough in the Early Stage of Phthisis.—A considerable number of patients give a history of repeated "colds" caught during several preceding winters or autumns; or of attacks of "grippe" which made them cough more or less violently, but they subsided under ordinary treatment. Owing to some neglect, the last attack has been per-

sistent, the cough aggravated, and could not be relieved by the remedy which helped them formerly. The cough in these cases is apt to be rather mild, consisting mainly in clearing the throat in the morning, and may not at all be productive of sputum; or small lumps of clear vitreous secretion from the nasopharynx may be brought out. Rarely mucopurulent material is eliminated, but it is usually devoid of tubercle bacilli at this stage. In many cases the cough is due to irritation of the sensory nerve endings of the vagus within the bronchial mucous membrane by the secretion derived from the tuberculous lesion. Here tubercle bacilli are found, as a rule.

These repeated attacks of "grippe," or "bronchitis," which subside during the summer, to return during the autumn and winter, and are easily managed by ordinary sedatives, often give the patient a false sense of security, and when told that the cough is of tuberculous origin he is loath to agree to it.

A careful examination of the throat is imperative in these cases. It must always be borne in mind that tuberculosis hardly ever begins with congestion of the rhinopharynx. When a cough can be rationally attributed to acute coryza or subacute rhinopharyngitis, and no signs are found while examining the chest, tuberculosis is only a very remote possibility. Even in those who are undoubtedly tuberculous, the fact that they have hyperplasia of the glands of the throat, that they are "lymphatics," points toward a mild, or even an abortive, course of the disease (see Chapter XXX).

Mild cough is to be differentiated from hysterical cough, which is very frequent at present when phthisiophobia is rampant. In fact, in many homes with tuberculous patients, notably after a consumptive dies in the house, most of the healthy members of the family cough, believing they are affected with the disease. Perhaps the best sign is that hysterical cough does not occur at night, when the patient is asleep, or during the day, when he is absorbed in some matter which interests him. I have seen patients who coughed persistently, cease coughing during the time they were engaged in an interesting conversation. In many cases the cough in incipient phthisis is annoying at bedtime, disappearing during the first hours of sleep, and reappearing during the early morning hours, often waking the patient, while after rising it may be intense until the chest is cleared. During the day it may be scarce or absent and provoked only by emotional disturbance, undue exertion, chilling the body, a dusty or smoky atmosphere, etc.

Paroxysmal Cough.—In many patients at the onset of the disease, or during its later stages, the cough is violent and paroxysmal; occurring in fits. When unproductive, it may be difficult to bear because it often increases in intensity during the evening, and keeps the patient awake during the night, causing pain in the chest, insomnia, and exhaustion. In others, the fits keep up for quite some time until a small piece of viscid mucus is expelled. The first thing these patients ask for is a remedy which will loosen the sputum. During such spells

vomiting may occur, or even involuntary evacuation of urine, especially in women with lacerated genitals. These paroxysmal explosions of cough are a frequent cause of hernia in men, especially in those suffering from fibroid phthisis.

Paroxysmal cough in phthisis is said to be due to ulceration of the trachea at its bifurcation. But it is also met with in cases of tracheobronchial adenopathy and adherent pleurisy. Its occurrence during periodical evacuation of pulmonary cavities will be discussed later on.

Patients suffering from fibroid phthisis, and those who have tuberculosis evolving in emphysematous lungs, suffer at times from severe paroxysms of cough. In these cyanosis of the lips and finger-tips, and bulging of the veins of the neck, are strong features during a paroxysm, and the suffering may be extreme. The violence of the cough is usually far out of proportion to the amount of sputum brought up. After the expulsion of a small lump of transparent mucus they feel relieved but exhausted, to be annoyed again at longer or shorter intervals. Nocturnal attacks are not uncommon.

I have observed similar paroxysms of violent cough in many cases of galloping consumption in which the lesions could not be localized; also in miliary tuberculosis with tubercles widely disseminated all over the lungs, and signs of pulmonary emphysema were elicited on physical exploration of the chest. Some authors believe that the violence of the cough may be responsible for the extensive dissemination of the tubercles by metastasis. But in many cases under my care the lesion finally localized itself, and the disease pursued the usual course of chronic phthisis, the paroxysmal cough disappearing, leaving the common cough encountered in the average case of the disease.

The Emetic Cough.—First described by Richard Morton at the end of the seventeenth century, the cough ending in vomiting, is quite frequently met with in the early stages of phthisis in various degrees of intensity. Some French authors, notably Paillard,[1] state that the *signe de Morton*, or the *toux émétisante*, as they call it, is met with to the extent of 50 to 60 per cent of all cases of phthisis. This has not been the case with the patients under my care. To be sure, vomiting may be seen in more than one-half the cases of tuberculosis at some period of the course of the disease, but not all vomiting can be considered the true emetic cough, as we shall soon show.

It has been stated that the cough of incipient phthisis often produces no expectoration, but vomiting. There are tuberculous patients who cough as soon as they eat, says Michel Peter,[2] there are others who cough because they eat; finally, there are others who, having eaten, cough, vomit, and suffer from cardiac palpitation. *This emetic cough is so characteristic that when whooping-cough and rhinopharyngitis in chronic alcoholics are ruled out, I place great reliance on it in doubtful cases*, and it has often helped me in making a positive diagnosis sooner

[1] La toux émétisante des tuberculeux, Paris, 1911.
[2] Leçons de Clinique médicale, Paris, 1879, **2**, 318.

than I could have made it without this symptom. But to appreciate its diagnostic significance it must not be confounded with vomiting of other origin which may occur in phthisis. It usually occurs in the following manner:

The patient has had his lunch, or dinner, with a variable appetite and feels rather satisfied, having no sensation of gastric disturbance, excepting perhaps some feeling of epigastric distention or mild dyspnea. But after the lapse of some time, from five minutes to an hour—an average of about twenty minutes—the patient, either without any warning at all, or feeling some irritation at the back of the throat, is seized with a paroxysm of cough which nearly chokes him; he feels as if he is unable to expel a piece of tenacious mucus which sticks in his throat. Finally he vomits out, in part or completely, the gastric contents which are in a variable state of digestion, according to the time they remained in the stomach. There is no sensation of nausea before the paroxysm, but the vomiting comes on suddenly during the coughing spell; a fact which differentiates this form of vomiting from other forms. When occurring for the first time the patient is alarmed, or may be inclined to attribute it to some dietetic indiscretion, but if it occurs repeatedly he is compelled to seek another cause. As soon as the vomiting ceases the patient usually feels greatly relieved, the sensation of gastric distention and the dyspnea disappear, and at times he may express a desire to eat again. After a time the patient learns prudence from experience—he knows that a heavy meal may bring about a fit of cough followed by vomiting.

During the course of phthisis there occur also other varieties of vomiting which cannot be classified under the heading of emetic cough. Patients who have been sufferers from chronic gastritis, dilatation of the stomach, and chronic alcoholism, often vomit; at times vomiting is provoked by cough. In the advanced stages of the disease vomiting, preceded by cough or not, may occur and in some patients it may be so pronounced as to preclude feeding. But these forms of the vomiting are not the true emetic cough. These patients usually suffer from symptoms of indigestion—nausea, furred tongue, foul breath, constipation, diarrhea, headache, etc. Examination usually reveals a dilated stomach, amyloid, or fatty degeneration of the liver, symptoms of tuberculous peritonitis, etc. Moreover, while the vomiting may occur after coughing, yet it is not invariably preceded by paroxysmal cough, occurs irregularly, not always after the ingestion of food, and there is no relief immediately after the vomiting. In alcoholics the vomiting is more apt to occur in the morning, and this is also the rule with those in whom the cough is due to chronic pharyngitis. In both these conditions, nausea, retching, etc., are common, while in the true emetic cough they are absent. *The emetic cough often occurs in the early stages of phthisis, in patients in whom the gastric functions are in good condition, is always preceded by spells of cough, always occurs at a certain time after the ingestion of food, is not preceded nor followed by sensations of*

nausea, giddiness, faintness, and retching. The reverse, vomiting and then coughing, is never observed.

This form of vomiting, or the emetic cough, is observed in practice in but a few diseases, namely, phthisis, whooping-cough, and in certain forms of pharyngitis, especially in alcoholics. Recently the writer has observed it in many cases of post-influenzal bronchitis and bronchiectasis. The fact that the thoracic glands are found enlarged in many of these cases explains its origin. When whooping-cough is excluded in a patient with an emetic cough, the pharynx is found to be in good condition, and post-influenzal bronchitis is not suspected, phthisis is at once to be thought of. If it persists, a diagnosis of tuberculosis may be made even in the absence of definite physical signs of the disease.

Some authors have been inclined to look at the emetic cough as a mechanical accident, comparable with that observed in whooping-cough. But it appears that this does not entirely explain this phenomenon. If the compression of the abdominal muscles and stomach were the sole cause, we should expect vomiting to occur during violent and prolonged asthmatic paroxysms. But I have never seen a patient suffering from asthma vomit after an attack of cough and dyspnea, and be relieved immediately after the gastric contents have thus been expelled.

As has been pointed out by Michel Peter, W. Soltau Fenwick,[1] Paillard, and others, the emetic cough appears to be purely a reflex phenomenon, due to irritation by the ingested food of the gastric ends of the vagus, and an abnormal excitability of the respiratory center. Hence, the slightest irritation of the gastric mucous membrane by particles of food is sufficient to produce a violent attack of reflex cough which can bring about vomiting in a mechanical manner.

Cough during the Advanced Stages of Phthisis.—With the advance of the disease the cough becomes more and more abundant, more productive, but easier, and less exhausting. After the formation of cavities, there is usually observed a diminution in the frequency of the cough, sleep is hardly disturbed during the night when the reflexes are in abeyance, and the secretions accumulate in the cavity. But in the morning, when compelled to empty the cavities of the secretions, there are fits of coughing lasting several minutes, perhaps an hour, and the patient feels relieved.

These patients, like those suffering from bronchiectasis, suffer from cough periodically when the excavations have been filled and need emptying. It may be influenced by posture—as soon as they change their position, the secretions overflow the bronchial tubes and must be brought out by cough, which does not cease until all has been discharged. Then there is relief for a variable time until the cavity is again filled. The patients usually learn from experience on which side to sleep if they want to have peace. It is not always on the healthy

[1] The Dyspepsia of Phthisis, London, 1894, p. 118.

side on which they can lie with more or less comfort, because, like in bronchiectasis, it depends on the direction of the bronchus, or sinus, which empties the cavity. Patients with pleural effusions also cough when changing their positions, but in their case the cough is usually dry, and is not instrumental in bringing up abundant sputum. For obvious reasons, patients cough more when lying down than when in the upright position. But in others sitting up in, or getting out of bed excites a paroxysm of cough and expectoration.

In some cases the cough at this stage is very severe and almost incessant, painful, and preventing rest day and night; actually exhausting. It is noteworthy that the severity of the cough does not altogether depend on the extent of the lesion in the lung, nor on the size and number of the cavities. Some will cough very little, although the lungs are extensively involved, while others, with limited infiltrations or excavations, cough severely.

The cough of tuberculous patients is often greatly influenced by various factors, of which the age and the emotional state are most important. Young adults cough, as a rule, more than old consumptives. In fact, a large proportion of old people suffering from phthisis hardly cough; they bring up large quantities of sputum without any effort. They are the patients who supply the material for those who describe cases which have been sick with tuberculosis for many years and never coughed. The psychic state of the patient also has a great influence. The nervous, irritable, and hysterical, cough more than the indolent and phlegmatic. The former class is also more apt to suffer from the emetic form of cough.

Diagnostic and Prognostic Significance.—On the whole, cough serves a very good purpose by drawing the attention of many patients to the condition of their lungs. *A person who never coughed, but "caught cold" for the first time after his eighteenth year, and as a result keeps on coughing for more than a month, is to be strongly suspected of being tuberculous,* even if there are no definite physical signs of a pulmonary lesion. The suspicion is fortified by a history of the absence of acute coryza during the first few days of illness, because simple bronchitis and "grippe" are almost always preceded or accompanied by rhinopharyngeal catarrh.

From the prognostic viewpoint cough is important because we meet cases with small pulmonary foci without much fever, anorexia, emaciation, etc., who would undoubtedly do well, but for a cough which is difficult to control. If violent, paroxysmal, and continuing for some time, the cough may be instrumental in extending the lesion, exhausting the patient, and thus aggravating the outlook. It also irritates the larynx, trachea, bronchi, and pulmonary parenchyma, and predisposes these organs to infection by metastasis of the bacilli. Violent fits of cough may also be responsible for spontaneous pneumothorax in cases in which the lesion is located superficially or subpleurally.

Kuthy and Wolff-Eisner[1] say that the most unfavorable prognosis is to be given in cases in which the patient coughs during both day and night; relatively more favorable is the outlook when he coughs during the day exclusively; more favorable when he coughs only mornings and evenings; and most favorable when he coughs exclusively in the morning.

Within certain limits cough also gives other prognostic hints. With each improvement in the local or general condition, the cough also improves or disappears, and with every recrudescence of cough we may find an extension of the process in the lungs, or some complication in the bronchi or rhinopharynx. *Occasionally we may note that the sudden disappearance of cough is a signal of some grave complication of phthisis, especially meningitis or peritonitis.* The same is at times seen in cases of severe ulcerations of the larynx, causing dysphagia, etc. The cough may be ameliorated, but the lesion in the lungs continues or extends and, combined with the exhaustion due to lack of nourishment, the end is not very far.

Hoarseness.—Changes in the timber of the voice may appear quite early in the disease without any tuberculous involvement of the larynx. The least provocation, such as changes in the weather, or prolonged speaking, may produce dysphonia, or a muffled voice, without any pain which, with the dyspnea preventing speaking continuously long sentences, may be quite troublesome.

In many cases the hoarseness is due to simple catarrh caused by local irritation of the larynx by the secretions while they are being eliminated from the lungs. In others, pressure of a tuberculous gland, lying between the trachea and the esophagus, on the recurrent laryngeal nerve causing adductor paralysis, is the cause. Reflex irritation of the superior laryngeal nerve may also be the cause of hoarseness. Often the hoarseness is due to tenacious secretions sticking to the vocal cords, and after coughing strongly they are dislodged and the voice is again normal. Congestion of the larynx caused by violent fits of coughing may be the reason for hoarseness.

It is thus evident that not all cases of hoarseness, or even dysphagia, are due to tuberculous ulcerations of the larynx. In fact, no diagnosis of the latter condition should be made without a careful and painstaking inspection of the larynx with a mirror by one who is competent and experienced.

EXPECTORATION.

Careful inquiry reveals in most cases that the cough preceded expectoration by several weeks or even months, and we must not unequivocally conclude that because the cough is unproductive we are not dealing with phthisis. Children before the sixth year never bring up any sputum at all, because they unconsciously swallow it, and most women do the same. I have met with cases in which urging women to expec-

[1] Die Prognosenstellung bei der Lungentuberkulose, Berlin, 1914, p. 219.

torate was of no avail. Many men are not much better in this regard and, for reasons of false delicacy, they swallow the sputum, especially during the early stages of the disease. In the advanced stage we may meet with the same condition when the emaciated patient is exhausted and hardly has any strength to rise, or turn around in bed and expectorate into the sputum cup.

With the advance of the disease the quantity of sputum eliminated increases, but I have met with cases showing extensive infiltrations of more than one lobe, without any substantial expectoration, and in some of these I have been convinced that they had not swallowed the sputum. It was merely an indication that the tubercles had not broken through a bronchus, or that the cavities were "dry."

Macroscopic Appearance of the Sputum.—There is nothing typical about the naked-eye appearance of the matter expectorated in early phthisis, although ancient clinicians gave detailed descriptions of typical tuberculous sputum. Perhaps the fact that they knew very little about early phthisis will account for their confidence in the gross appearance of the sputum in this disease.

In the early stage we find that the sputum is scanty; at times it is altogether absent. Kuthy found that in 49 per cent of cases in the first stage, 15.4 per cent of the second stage, and 12 per cent of the third stage, sputum was altogether absent. What is usually brought up in the early stage is viscid mucus, occasionally with some dark specks; it is often frothy and floats on water, hardly differing from the expectoration in bronchitis.

With the advance of the disease the sputum becomes thicker, although it remains glassy or transparent for some time, but yellow streaks are to be seen, indicating that it is assuming a purulent character. Later its appearance and consistency change: It becomes mucopurulent, and finally purulent, indicating that softening of lung tissue has taken place and the necrotic parts are being eliminated. The purulent character of the expectoration is judged by the yellow, yellowish-green, or green color it assumes. Pure purulent sputum, without froth, is mostly seen in cases in which an abscess, an empyema, or pyopneumothorax has broken through a bronchus.

In the far-advanced stage of the disease the sputum is usually dark gray, or greenish in color, made up of roundish balls which float around like islands in the fluid mucus or saliva or, when thicker in consistency, sink down to the bottom of the receptacle, where it settles in disk or coin-shaped masses which keep apart and do not coalesce. This is the *nummular sputum* of old physicians which had erroneously been considered pathognomonic of phthisical excavations. At times whitish, cheesy masses, derived from broken-down tubercles, are seen scattered within this sputum.

This sputum is usually odorless, but at times it acquires a very disagreeable, sweetish, but nauseating odor, especially when retained within the chest by narcotic drugs, or weakness of the patient. Fetid

and offensive sputum is exceedingly rare in phthisis. Whenever it is met with in a case of undoubted phthisis we should look for complicating pulmonary gangrene, which occurs at times. More commonly fetid sputum is indicative of some non-tuberculous pulmonary disease, especially abscess and gangrene of the lung, malignant disease of the bronchi or lung, etc.

In tuberculosis the sputum is usually salty in the early stages of the disease, but later it often acquires a sweetish, sickening taste.

Very often this sputum, derived from tuberculous cavities, when allowed to stand in a vessel for some hours separates into three layers —an upper frothy layer; a middle thin serous layer; and a lower layer consisting of thick plugs of pus. This is characteristic of excavation but is not necessarily of tuberculous origin. Bronchiectasis and chronic bronchitis with copious expectoration may also be productive of sputum which separates on standing. However, in the former the lines of demarcation between the layers are not so distinct, but one passes into the other by slow gradations. In rare instances calcareous masses, "stones," may be found in sputum derived from tuberculous cavities.

There are cases of advanced chronic phthisis with scanty, or even without any expectoration, especially those of the type of fibroid phthisis, or with emphysema, although they have periods in which the expectoration is quite profuse. The expectoration decreases in quantity when the cavities "dry up" during the process of healing, and in other types of cases when the concomitant bronchitis disappears. With but few exceptions, scanty expectoration speaks for a favorable outlook, provided the temperature is normal and the cough is also absent or mild. On the other hand, copious expectoration *per se* is not always an unfavorable sign. It is an indication of excavation, bronchitis, or bronchiectasis which are not infrequent in phthisis. In the latter cases the sputum may show a tendency to collect and be expelled at intervals in very large quantities—mouthfuls—without any effort, and may also be influenced by posture.

During hemoptysis the material expectorated is sanguineous in various degrees, corresponding to the severity of the bleeding, and for a few days after the cessation of the active hemorrhage the sputum contains dark clots derived from the blood that has coagulated in the excavations or the bronchi and is being slowly eliminated. The sputum may have a reddish or chocolate tinge without distinct hemorrhage, and even viscid, rusty sputum characteristic of pneumonia is at times encountered in phthisis. Inasmuch as this is, as a rule, seen during an acute exacerbation of fever, etc., I am inclined to account for it, in many cases, by intercurrent pneumonia. In the terminal stage of advanced cases I have often seen thin, watery sputum, dark brown in color, with numerous air bubbles—prune-juice sputum—which is an indication of pulmonary edema. Green sputum is at times met with, and is usually ascribed to the implantation of the *Bacillus pyocyaneus*

In cases in which a pyopneumothorax communicates with a bronchus, as well as when an empyema breaks through a bronchus, the sputum may be distinctly purulent, and I have seen cases in which the empyema was thus cured, though the tuberculous process went on its course.

EXAMINATION OF THE SPUTUM.

Collection of Specimen.—In cases of suspected phthisis the sputum gives important information which is often of more value than all other diagnostic methods for this disease taken together. This is especially true of the microscopic examination.

It is important, especially in cases with scanty expectoration, that the specimen of sputum for examination should be properly collected. The patient must be warned that what we want is material that has been coughed up from beneath the glottis, and not what has been hawked out from the rhinopharynx, or saliva. A clean, wide-mouthed bottle is the best receptacle, and it should be tightly corked. The one used by the Health Department in New York City is excellent. In cases with scanty expectoration, a twenty-four-hour specimen is desirable, but with others the quantity coughed up during the morning on rising is sufficient. Fresh sputum is best, but putrefaction does not interfere with the appearance of the bacilli under the microscope.

It must be emphasized that really active cases of tuberculosis with persistently negative sputum are rare. Most of these cases, if examined repeatedly, will show the presence of tubercle bacilli in the sputum. In my wards at the Montefiore Hospital we often find that these "closed" cases show the presence of bacilli after several examinations of the sputum. In some it takes as many as twenty microscopic examinations to find one positive. But it is doubtful whether a patient who shows persistently negative sputum is in fact sick with tuberculosis requiring treatment, and I have been under the impression, based on good evidence, that the sanatoriums which have as many as over 50 per cent of "sputum negative" cases have an enormous proportion of non-tuberculous cases within their walls. I doubt whether more than 10 per cent of these "sputum negative" cases are tuberculous in the clinical sense.

On the other hand, it is wrong to consider a case as not contagious because the sputum is negative. We are of late beginning to realize that the sputum is not the only vehicle in which tuberculosis is transmitted from the sick to the well. This point has been discussed in detail elsewhere in this book.

Microscopic Examination.—In the very early stages of active phthisis tubercle bacilli are more often absent than present in the sputum, and it is only after softening of tubercles has taken place and the diseased focus opens into a bronchiole that they can be found. In general, it may be stated that severe cases show large numbers of bacilli, but there are many exceptions. In fact, in acute pneumonic phthisis bacilli are,

as a rule, lacking. The absence of bacilli is therefore not conclusive proof of the non-tuberculous character of a case, because we meet with undoubted cases of active and progressive tuberculosis, proved by subsequent autopsy findings, in which no bacilli were discovered throughout the course of the disease. In general, it may, however, be stated that these "closed" cases of tuberculosis run a more favorable course. On the other hand, in acute miliary tuberculosis, tubercle bacilli are discovered in the sputum in exceedingly rare instances.

In early phthisis in which it is difficult to obtain sufficient sputum for examination, the administration of iodides, 5 grains three times a day for a couple of days, may increase the amount of expectoration. We may, in some cases, also administer an opiate in the evening with a view of retaining the sputum during the night, so that it may be brought up in the morning on rising. In children, swabbing the throat with some gauze, as suggested by Holt, may yield a specimen for examination, though in my hands it has invariably failed.

Technic.—The examination is best and most rapidly accomplished by the Ziehl-Neelsen, the Gabbet, or the Hermann methods, which have survived numerous modifications introduced during recent years. Details have been given on page 18.

With a platinum-wire loop a cheesy or mucopurulent particle is picked out and spread over a perfectly clean cover-glass in a thin, uniform layer. It is even better that a small amount of sputum should be spread between two cover-glasses which are drawn apart. The cover-glass is dried in the air, or over a Bunsen burner at some distance from the flame. When dry, it is "fixed" by passing it three or four times through the flame. Some carbol-fuchsin solution is put on the specimen which is picked up with a Cornet forceps and held over the flame for about three minutes or more until it steams. It is then decolorized in a 10 per cent solution of nitric acid, or a 10 per cent solution of sulphuric acid and washed in 60 per cent alcohol, until it is faintly pink or completely colorless, when it is counterstained with an alcoholic solution of methylene blue, washed in water, and dried between filter paper.

With Gabbet's method the staining with carbol-fuchsin is the same as above, but the decolorization and counterstaining are done together by placing the specimen in Gabbet's solution (see p. 19).

The Hermann stain is also easy; it consists in: (*a*) Crystal violet, 3 per cent in alcohol; (*b*) ammonium carbonate, 1 per cent solution in water. Mix one part of solution *a* with three parts of solution *b* just before using. Steam as above, decolorize with 10 per cent nitric acid, wash in alcohol, and counterstain with Bismarck brown. At times this method will reveal bacilli when the above have failed.

These methods will disclose the bacilli in the vast majority of cases, but they fail at times because of the small amount of sputum available, or the small number of bacilli present in the specimen, or the selection of a particle of sputum with the platinum loop which does

not contain any bacilli. To obviate these sources of error there have been devised new methods which liquefy the sputum, digest all the cells and bacteria which may be present, excepting the tubercle bacilli, which can be centrifuged and be examined microscopically, and may even be used for cultural purposes, or for injections into animals. The antiformin method is at present the best and simplest available for the purpose.

The Antiformin Method.—This method, devised by Uhlenhuth and Xylander, and modified by others, consists in mixing the sputum with antiformin—a strongly alkaline mixture of sodium hypochlorite, equivalent to 5.68 gms. available chlorine; sodium hydroxide, 7.8 gms., and sodium carbonate, 0.32 gm.—used by brewers in the disinfection of their fermentation vats and tubes. When properly diluted and mixed with sputum, there is a strong liberation of gas, the insoluble organic matters, as well as bacteria, are destroyed, excepting hair, fat, wax and cellulose, and acid-fast bacilli, the vitality and staining reactions of which remain unchanged. The resulting yellowish solution is a homogeneous mixture with a flocculent sediment. Because it has a fatty capsule the tubercle bacillus remains intact while all other microörganisms are rapidly destroyed.

Of the various modifications of Uhlenhuth's original method, the one devised by Boardman[1] is the most serviceable. It consists in:

1. Place the entire twenty-four-hour sputum in a conical settling glass; if the amount is excessive it is perhaps better to use only 15 to 20 cc.

2. If the specimen is thick, add an equal volume of distilled water. Less tenacious specimens do not require so much dilution.

3. Add an amount of antiformin equal to one-fourth the volume of the diluted sputum; in other words, sufficient to make a 20 per cent solution.

4. Stir thoroughly, thereby breaking up the masses of mucus and greatly hastening complete solution.

5. Allow to stand till solution appears homogeneous. It should now be watery in consistency and pale yellow in color; if necessary, more water or more antiformin should be added and digestion allowed to continue. This will usually require from a few minutes to an hour, but may be allowed to continue for days with no resulting harm to the tubercle bacilli.

6. Add an equal volume of 95 per cent alcohol. By this procedure the specific gravity is reduced from about 1.030 to below 1; thereby not only hastening sedimentation, but making it more complete.

7. After stirring, allow to stand till sedimentation is complete. This will occur in from two to four hours, but a period of twelve to twenty-four hours is recommended. During this sedimentation it may be necessary to gently turn the vessel to dislodge little particles of sediment which may be adhering to the sides of the vessel.

[1] Johns Hopkins Hosp. Bull., 1911, **22**, 269.

8. Pour off the clear supernatant fluid.

9. Make a smear from the sediment on a glass slide, using some of the original sputum to aid in fixing the smear. This is best done by making a smear from the sputum before antiformin is added and afterward spreading the sediment from the sputum-antiformin mixture on the same slide. Stain in the usual way.

There are many modifications of this method which do not require twenty-four hours for execution. Loeffler's modification, which takes but ten minutes, is the best:

A certain quantity of sputum (10 to 20 cc) is mixed with an equal quantity of 50 per cent aqueous solution of antiformin and boiled over the flame. Rapid liquefaction is observed. To each 10 cc of the mixture, 1.5 cc of a 10 per cent alcoholic solution of chloroform is added. After stirring for some time the solution is centrifuged for about fifteen minutes. The disk which forms on the surface of the chloroform contains the tubercle bacilli, and is to be pipetted, fixed with egg albumen, and stained in the usual way.

Theophile Raphael's[1] modification of Greenfield and Anderson's[2] method is even more reliable and time-saving. The procedure is as follows:

Five cc of sputum, to which have been added two volumes of 1 per cent solution of sodium carbonate in 1 per cent phenol, are shaken for ten minutes and then autoclaved for twenty minutes at fifteen-pounds pressure. Following this, the material is centrifuged for ten minutes, the supernatant fluid decanted, and smears are made and stained from the sediment. The following reagent is employed: Equal parts of 15 per cent sodium hydroxide and Labarraque's solution. Five parts of sputum with one part of reagent are shaken for fifteen minutes, incubated for one hour, and then centrifuged for fifteen minutes. The supernatant fluid is decanted and the sediment washed by centrifuging for fifteen minutes with distilled water. Smears are made and stained from the sediment.

The great importance of the antiformin method lies in the fact that it exerts a destructive action on all cells and microörganisms excepting the acid-fast rods which may then be found microscopically. But soon after its introduction it was found that acid-fast rods which are not pathogenic, and which are often found while looking for tubercle bacilli, may escape destruction by the antiformin, thus causing mistakes. Especially was the question whether the smegma bacillus is dissolved by this agent important. In a recent investigation of this problem by von Spindler-Engelsen,[3] she found that the smegma, the timothy-hay bacillus, the butter bacillus, etc., are dissolved by 15 per cent of antiformin in thirty minutes. The human and the bovine types of tubercle bacilli were not affected with a 50 per cent antifor-

[1] Jour. Am. Med. Assn., 1920, **75**, 245.
[2] Lancet, 1919, **2**, 423.
[3] Centralbl. f. Bakteriol., 1915, **76**, 356.

min solution for four days. Under the circumstances it appears that the pathogenic bacteria may be discovered with the aid of this method. It is, however, important that a fresh solution of antiformin should always be used, because a weak and old solution may leave the non-pathogenic bacteria and thus lead to error.

Much's Granules.—As has already been stated, there are cases of pulmonary tuberculosis in which no acid-fast bacilli can be discovered in the sputum by any method, and Much has shown that they are due to a certain kind of bacilli which have lost their acid-fast property, but are Gram-positive and they retain their virulence. According to some authors these Much granules are almost always found in cases of fibroid phthisis, chronic bronchitis, emphysema, bronchiectasis, etc., in which acid-fast bacilli are very rarely discovered (see p. 19). Much found them in cases of cold abscess.

As to the reasons why the bacilli lose their acid-fast properties, there is no agreement. It also appears that the proportion of cases in which they are found varies with different observers, some having detected them in as many as one out of eight sputa, while others in less than 2 per cent. Much gives several methods for staining these granules. The following is the most suitable:

A very thin smear is made of the sputum and allowed to remain for twenty-four to forty-eight hours in a methyl-violet solution (methyl-violet, 10 cc of a saturated solution, in 100 cc of a 2 per cent watery solution of carbolic acid) at 37° C. temperature; or it may be stained by boiling for a few minutes over the flame. Wash and stain for one to five minutes with Gram's iodine and put for one minute in a 5 per cent nitric acid solution, then in a 3 per cent hydrochloric acid solution for ten seconds, and finally complete the decolorization by placing it for a few seconds in acetone-alcohol (equal parts of acetone and alcohol). Wash and dry.

Prognostic Value of Microscopic Findings.—The interest displayed by many patients, as well as by physicians, in the number of bacilli found in a specimen of sputum examined with a view to drawing prognostic conclusions is unjustified. There are cases which show but few bacilli in each specimen, yet they run a very acute and progressive course, while others with numerous bacilli pursue a slow, chronic course, terminating in recovery. Especially is this seen in senile phthisis, in which the number of bacilli expectorated is enormous and we may, in fact, speak of pure cultures; yet these "bacilli carriers" live on for years with comparative comfort. Of course, in such cases we may deal with a small ulcerating cavity in the lung which offers good opportunities for the growth of bacilli, but the fibrous capsule prevents the extension of the lesion.

The number of bacilli in the sputum fluctuates from day to day, evidently depending to some extent on the bit of sputum we happen to pick up with the loop. On the other hand, the complete absence of bacilli from the sputum for several weeks, coupled with improve-

ment in the general condition of the patient, is undoubtedly a favorable sign. But many chronic cases, especially fibroid phthisis, are always "closed"—bacilli are scanty or absent. With modern methods of antiformin examination of sputum the number of active, but "closed" cases has been reduced very much. In addition to the tubercle bacilli, various other microörganisms are frequently found in the sputum of tuberculous patients. They are usually saprophytes, and have no influence on the symptomatology and course of the disease. In contradistinction to this "passive mixed infection," we meet with "active mixed infection," in which the organisms, pneumococci, Pfeiffer's bacilli, streptococci, staphylococci, etc., are responsible for complicating processes. These are discussed elsewhere in this book.

Inoculation.—In cases in which it is very important to decide the problem whether we are dealing with active tuberculosis, and the bacilli are too few in number to be demonstrated by the ordinary microscopic methods, or after concentration of the sputum, inoculation of a susceptible animal often leads to a decision. In this regard, it must always be remembered that the susceptibility of ordinary laboratory animals to the tubercle bacilli varies greatly. The white mouse and the rat are relatively insusceptible. So is the rabbit to the human type of the bacillus, but it is very susceptible indeed to the bovine type. Hence, inoculation of the rabbit is a substantial aid in the differentiation of the human from the bovine species. The guinea-pig is exceedingly susceptible to both mammalian types of the tubercle bacillus, and is the animal ordinarily used for inoculation with material suspected of harboring tubercle bacilli.

If the material to be inoculated into the guinea-pig is very badly contaminated with other bacteria, it is best to subject it to the action of antiformin; but this should not be carried too far, lest the virulence of the tubercle bacilli themselves be seriously impaired. The material is suspended in sterile salt solution (though sometimes solid blocks of tissue or sputum are placed under the skin and the wound protected with cotton and collodion) and injected intraperitoneally, or subcutaneously, or better, perhaps, in both sites. The intraperitoneal injection gives quicker results, but there is greater possibility of losing the animal through a non-tuberculous infection before tuberculosis can develop. An autopsy performed four to six weeks after intraperitoneal injection, if tubercle bacilli were present in the suspected sputum, will show widespread tuberculous lesions of the peritoneum, the omentum, the retroperitoneal lymph glands, the spleen, the liver, and sometimes the lungs. Examination after subcutaneous injection of the suspected material, when positive, shows a caseating lesion at the site of the inoculation, also similar lesions in the regional lymph glands, liver and spleen. In addition to caseous lesions of the viscera, large areas of necrosis are often seen. One should never be content with the gross appearance of the lesions, but should always stain sections for Koch bacilli, which are usually present in large numbers. By removal

of enlarged glands with the animal etherized, the bacilli can often be demonstrated in section after ten to fourteen days.

There are, however, in rare instances, cases in which it is of great importance to ascertain the presence or absence of tubercle bacilli in the sputum sooner than in six or eight weeks. Some have suggested that after the suspected material has been injected into the abdominal wall or the peritoneum, the animal should be tested at frequent intervals with tuberculin. A positive reaction clears up the case (Römer and Joseph[1]). F. Gratz[2] has used the intracutaneous method. He inoculated 1000 guinea-pigs and then applied the intracutaneous tuberculin test and found that in ten or twelve days after the inoculation of the infectious material a positive diagnosis may be made. Martin Jacoby and N. Meyer[3] suggest that the sputum be injected into a guinea-pig and about fourteen days later 0.5 cc of tuberculin should be injected subcutaneously. If the sputum contains tubercle bacilli and infects the animal, it will die from anaphylactic shock within a few hours.

But these methods are not infallible. Selter[4] shows that a positive reaction in an inoculated guinea-pig indicates that infection has taken place, while a negative result does not prove the contrary. The autopsy alone is conclusive. Many guinea-pigs inoculated by Selter with small doses of virulent bacilli were found to give negative results to the intracutaneous test, while the autopsy revealed marked tuberculous changes in various organs.

It must also be mentioned in this connection that guinea-pigs are often tuberculous spontaneously. Many authors have reported that they found tuberculous lesions in these animals. Sir Almroth Wright, Frank J. Clemenger and F. C. Martley[5] point out that great difficulties are encountered in obtaining guinea-pigs free from pseudotuberculosis; a large proportion of the animals were found affected with various forms of this disease. In a lot purchased from a guinea-pig fancier who bred his animals exclusively for purposes of exhibition, and which were young and, from all outward appearances, perfectly healthy, a point was made to autopsy with great care each of the animals of this lot that had been killed for the purposes of securing fresh serum for complement, and pseudotuberculous lesions were found in every one of them. "The amazing point about these infections with pseudotuberculosis is the large amount of vital organs which can be involved in the local process and yet permit the animals to live in apparent health." The possibility of error while utilizing guinea-pigs for diagnostic experiments is manifest.

Elastic Fibers.—Before the discovery of the tubercle bacillus great stress was laid on the presence or absence of elastic tissue in the

[1] Beitr. z. Klin. d. Tuberk., 1909, **14**, 1.
[2] Ibid., 1916, **36**, 99.
[3] Ibid., 1911, **20**, 263.
[4] Deutsch. med. Wchnschr., 1916, **42**, 77, 283.
[5] Senate Document No. 453, Washington, 1916.

sputum in the diagnosis of tuberculosis, but of late this is only rarely looked for. It is, however, a simple thing to find elastic tissue when present in the expectoration, and it is of immense diagnostic significance because it can be found in over 90 per cent of tuberculous sputa.

The presence of elastic fibers in the sputum is an indication of destruction of lung tissue and it may be found in the very early stages of the disease, because chronic tuberculosis is a destructive process, and small excavations may be found quite early, and the elastic fibers are not destroyed during the caseous degeneration which liquefies the pulmonary tissue. They are also found in gangrene, abscess, syphilis, and infarction of the lung, so that when the latter can be excluded, they may greatly assist in the diagnosis of doubtful cases of tuberculosis.

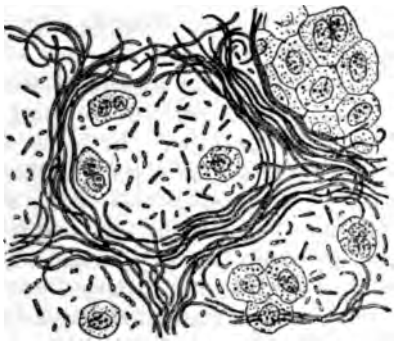


FIG. 33.—Elastic fibers in the sputum. (v. Jaksch.)

Technic.—A small amount of the thick, purulent portion of the sputum is pressed into a thin layer between two pieces of plain window-glass, 15 x 15 cm. and 10 x 10 cm. The particles of elastic tissue appear on a black background as grayish-yellow spots, and can be examined *in situ* under a low power. Or, the upper piece of glass is slid off until the piece of tissue is uncovered, when it is picked out and examined on a slide, first with a low and then with a high power (Simon).

A simpler method is the following: A bit of purulent sputum and a drop of 10 per cent solution of sodium or potassium hydroxide are placed between a cover-glass and a slide and examined with a moderate power under the microscope. The elastic tissue is to be looked for especially at the border of the preparation.

If the fibers are scanty they may not be found in this way, and the following method may reveal them: The sputum is boiled with a 10 per cent solution of KOH and well stirred during the boiling. When a homogeneous mixture is obtained, it is diluted with four times as much water, well shaken, and allowed to stand in a conical glass, or centrifuged. The sediment contains all the elastic tissue, which may be found under the microscope.

The different methods of staining elastic tissue are not necessary because either of the above methods is sufficient for diagnostic purposes.

Cytology of Sputum.—Various attempts have been made to assign diagnostic and prognostic significance to the cytology of tuberculous sputum, especially to the leukocytes and lymphocytes, but without avail. Nothing diagnostically important can be learned from a study of the white blood cells in the sputum, so far as we know at present.

Chemical Examination.—The chemistry of the sputum in pulmonary tuberculosis has not yielded any important diagnostic or prognostic data, excepting the albumin reaction, which is of great value in doubtful cases and is often of assistance when the microscope fails to reveal tubercle bacilli. Sputum with a positive albumin reaction can be found in tuberculosis and also in cases of pulmonary emphysema with cardiac dilatation, pneumonia, pleurisy with effusion, etc., but never in uncomplicated bronchitis.

A positive albumin reaction is not always decisive of tuberculosis, but the negative outcome, when persistent during several examinations, undoubtedly excludes phthisis.[1] In some cases of advanced tuberculosis, especially fibroid phthisis, the albumin reaction is negative, but in such cases the diagnosis is only rarely a problem. It also appears that with the improvement in the condition of the average patient, the amount of albumin in the sputum decreases and finally it disappears. It is thus of prognostic value.

Technic.—The albumin test is made as follows: A 3 per cent solution of acetic acid is added to the sputum, which is then thoroughly shaken. During ten or fifteen minutes the bottle is allowed to stand, and repeatedly shaken during this time. It will be observed that the mucus is coagulated by the acetic acid, and when it is then filtered through paper into a test-tube, the filtrate appears as a clear fluid. Occasionally all the mucus is not coagulated with the first attempt, and this is easily ascertained by adding a drop of acetic acid to the filtrate, which in such cases again shows flocculi collecting as a precipitate. The process is then repeated until a clear filtrate is obtained. The clear fluid is next boiled over a Bunsen burner or an alcohol lamp, and while boiling some crystals of common salt, or a concentrated solution of sodium chloride are added.

If albumin is present, there results a cloudiness, or a curdy precipitate which, on standing, settles to the bottom of the tube. Roughly speaking, the amount of the precipitate gives an idea of the amount of albumin present. The most important precaution to be observed is that nothing but a curdy precipitate should be considered as positive, because the presence of mucus, which the acetic acid does not always completely dissolve, may also give a cloudy precipitate on boiling. But this precipitate is not curdy, nor does it settle on standing. Of course, any other test for albumin may be applied to the filtrate, but the above gives satisfactory results.

[1] Fishberg: Med. Press and Circular, 1912, **94**, 352; Arch. of Diag., 1912, **5**, 220.

CHAPTER IX.

FEVER AND NIGHTSWEATS.

FEVER.

FEVER is one of the first symptoms of active phthisis—perhaps the first. It does not run a characteristic course in every case like that in malaria, pneumonia, or typhoid fever; in fact, its polymorphism is noteworthy. Yet it is of immense diagnostic and prognostic value. Some authors state that the fever in incipient tuberculosis is invariably due to some complication. But the febrile reaction after the administration of tuberculin, as well as in acute miliary tuberculosis, shows clearly that this view is incorrect. All the available evidence combines to prove that it is due to absorption of the poisons produced by the tubercle bacilli, though it may be modified by mixed infection. The fever is engendered mainly by the increased production of heat—the result of complex biochemical processes having their origin in the struggle of the organism with the bacilli; the body summoning its defensive forces against the toxins produced by the metabolic processes of the bacilli and decaying tissues which stimulate the heat regulating center. *In evaluating the significance of fever in tuberculosis, it must be borne in mind that it is not the cause of the disease, but a result of its activity.*

Fever is present in nearly all cases of active disease. In the later stages, especially in fibroid phthisis, we often meet with afebrile periods of shorter or longer duration, but with each exacerbation of the disease, with each extension of the process in the lungs, there is always a pronounced rise in the temperature which should be studied if the evolution of the case is to be followed.

Thermometers.—*The reason why there are found so many apyretic cases of phthisis is mainly faulty technic in taking the temperature, especially defective thermometers.*

The clinical thermometer is an instrument of precision, and when used for the purpose of ascertaining the temperature in incipient phthisis, in which 1° is occasionally of immense importance in diagnosis and prognosis, it must be accurate. It is, however, a well-known fact that, despite the certified accuracy of each instrument, simultaneous observations made on a single patient with two instruments often disclose a difference in readings of 0.75° to 2.° The simultaneous immersion of two dozen thermometers in a bath of warm water disclosed that the readings varied from 98.2° to 101.6° F.; another similar batch of higher-priced thermometers in another bath showed

variations of temperature between 98° and 105.4° F.[1] "Certified" thermometers in this country are not much better. Bray[2] reports that out of a series of 83 certified thermometers tested in a water-bath, 17 showed a variation of 0.3° to 0.6° F. Comparative rectal readings approximated closely the discrepancies shown in the water-bath. The presence or absence of fever, when such thermometers are used to ascertain it, depends on the instrument which the physician happens to possess and not at all on the condition of the patient. Under the circumstances, it is clear that when searching for fever in tuberculous patients or suspects, the instruments must be reliable and of tested accuracy, otherwise grave diagnostic mistakes of omission or commission are likely to occur.

Technic of Taking the Temperature.—After having a good thermometer, we must exercise great care in the method of taking the temperature. I have been so often misled by readings taken in the axilla, sometimes finding it as much as 3° below that recorded in the rectum, that I now completely discard it. And, strange to say, I meet with no patients who refuse to take their temperature per rectum. It has been found that in some cases the temperature in the axilla is higher on the affected side and urged as a good sign of phthisis, but it is so rare that it may be disregarded.

The mouth temperature is also unreliable to a certain extent. Here it is influenced by the temperature of the external air which must be inhaled now and then, especially by patients suffering from nasal obstruction. The part of the instrument outside the lips, and at times also the part within the mouth, are chilled by the external air, more often in dyspneic patients. The instrument must be left in the mouth at least seven minutes, and it often takes at least ten minutes before the mercury rises to the highest point, even with the so-called "minute thermometers." On the other hand, in patients suffering from stomatitis, the local temperature may be much higher than that of the blood. The temperature in the mouth should also not be taken immediately after meals, after taking hot or cold drinks, after washing the mouth or brushing the teeth, etc. Many patients are unable to keep the thermometer properly beneath the tongue, all surrounded by buccal mucous membrane, and avoid breathing through the mouth, or talking for five to ten minutes.

It appears that the majority of physicians in sanatoriums are in favor of oral readings because they are dealing with patients who practically always associate in groups and cannot use the rectal method unless they retire to their rooms for the purpose several times a day. This drawback does not hold with bed-ridden patients, and also with the average clientele in the city. In fact, I found that suspects, who keep at their work while under medical observation, prefer the rectal method which they take in the lavatory and thus obviate observation

[1] Lancet, October 4, 1913; November 8, 1913, p. 1342.
[2] Am. Jour. Med. Sci., 1915, **149**, 838.

by others. In my hospital work also, there is no trouble in taking rectal temperature in walking patients.

That the rectal method is superior and less likely to mislead is now acknowledged by all who have given both methods a trial. In the rectum or vagina the instrument is on all sides surrounded by mucous membrane, holding it in place as long as necessary and giving reliable readings. It has been found that the rectal is almost invariably 0.5° to 1° F. higher than the mouth temperature (Fig. 34). Some writers state that after exercise the local temperature in the rectum rises, while it remains normal in the blood, and for this reason they prefer mouth readings. But this again may be explained by the greater exactitude of the rectal temperature. It is needless to add that the instrument is to be left in the rectum sufficiently long to obtain the maximum reading. In my instructions to patients and nurses, I tell them that I do not know of any one-minute thermometers, and all are to be left *in situ* at least five minutes.

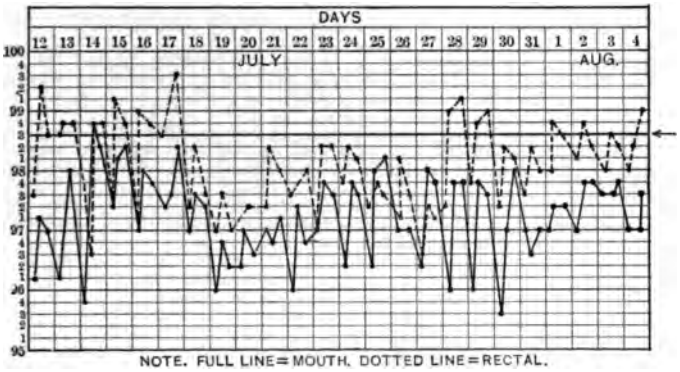


FIG. 34.—Comparative oral and rectal readings of temperature. (Bray.)

Frequency of Taking the Temperature.—The habit of many physicians of taking the temperature when the patient visits them and recording it as normal, or elevated to a certain degree, is altogether wrong. In early, or doubtful, cases taking three readings a day may be misleading, at times, because rises in temperature which occur late at night, or early in the afternoon, and are short-lived, may thus be overlooked and the patient pronounced free from fever. For reasons which will soon be evident, we must, in incipient cases, have a record of the temperature taken every two hours, and this is best recorded by plotting a curve on a chart which shows graphically any hypothermia or hyperthermia.

Intelligent patients may be entrusted with a thermometer, provided they are trained in reading it correctly, which can be done in a few minutes. I have had patients who kept records of their two-hourly temperature for weeks and, for obvious reasons, more conscientiously

than the average nurse. Many have done it without leaving their occupations by simply going to the lavatory every two hours for five minutes.

The Normal Temperature.—It may be stated that the normal temperature in children is not a constant value. It is subject to such oscillations during perfect health, that any average which has been fixed by various authors is only arbitrary. The slightest disturbance in health is likely to increase the temperature in the child to a greater degree than in the adult. Many clinicians consider a temperature of 100° to 101° F. normal in a child, unless there are symptoms of disease. But with advancing age the temperature becomes more and more settled, so that in adults it is subject to lesser oscillations, unless raised or depressed by disease.

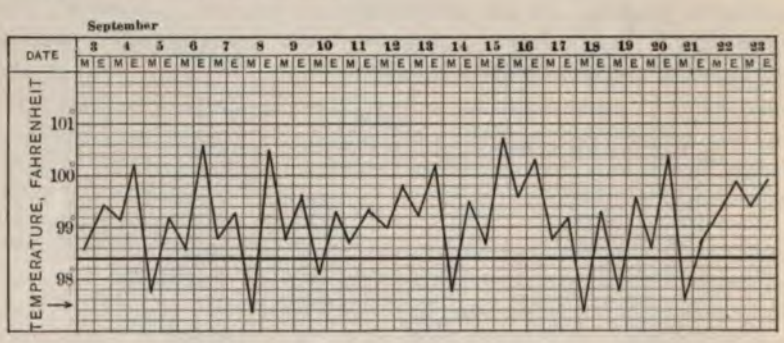

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FIG. 35.—Fever in incipient tuberculosis showing marked subnormal temperature in the early morning hours. Temperature taken twice daily.

As an arbitrary guide for the clinician it may be assumed that a temperature of 98.6° F. when taken by mouth, and 0.5° higher when taken by the rectum, is normal. But even this shows striking diurnal variations in normal individuals. During the early morning hours, before the individual leaves his bed, it is slightly subnormal by 0.5° to 1°; but it rises to normal soon after rising, and keeps quite steady during the day. Bardswell and Chapman[1] found an average for waking hours 98.5° F., and for sleeping hours 97.2° F., taken by mouth, which is in agreement with the observations of most physicians.

There are, however, individuals in whom the temperature is lower than the above average and in whom a physiological normal temperature should be considered febrile. This is occasionally seen in tuberculous patients with subnormal temperature; when the thermometer registers 99° F. they present symptoms of fever, such as flushing, hot skin, headache, etc.

Normally the temperature is elevated in persons after exercise, and in some even after a hearty meal. In women it may be higher by 1° or

[1] British Med. Jour., 1911, **1**, 1106.

2° before, or during menstruation. But the elevation after exercise is, in the healthy individual, evanescent; within one-half to one hour it sinks again to normal.

Other influences which should be mentioned are the emotional state of the individual. Particularly in women, excitement may raise the temperature 1° to 2°. Where there is a question of tuberculosis, the excitement attending the taking of the temperature may be effective in raising it, as I have seen in several cases, and we must be very careful in making a diagnosis of incipient phthisis on the thermometrical readings alone in emotional women. Frank B. Wynn,[1] in a recent study of the subject, found that psychic influences very frequently raise the temperature of healthy individuals. Observations made on two series of persons under circumstances of considerable nervous tension, suspense, and anxiety, such as physical examination during the selective draft, students and nurses taking State Board examinations, etc., showed distinct elevation of temperature in a large proportion of cases, the degree of elevation varying directly with the gravity of the situation facing the individuals. In fact similar observations have been made on animals. The frightened rabbit, tied to the operating board, shows elevation of temperature, as has been found by Moore.[2]

In some people who work during the night, and sleep during the day, the variations in temperature mentioned above are said to be reversed.

In evaluating thermometrical findings in suspected incipient phthisis, we are on safe ground when we consider the normal temperature during the day in a person who works, or walks around, as 99° F., when taken per rectum, and 0.5° to 0.75° lower when taken by mouth. It may be 0.5° to 1° lower in the morning before rising, and 0.5° higher in the evening after a heavy meal, or after a hard day's work. Distinct variations from these figures demand explanation, and if no other cause is found, tuberculosis is to be considered as the possible cause.

Fever in the Incipient Stage.—When taken with due precautions it will be found that *a subfebrile or febrile temperature is characteristic of the evolution of active phthisis even in the incipient stage, and that the absence of fever excludes active disease.* The afebrile cases of phthisis mentioned by physicians are mostly the result of faulty technic in taking the temperature. Evanescent rises are overlooked. Moreover, in these cases the instability of the temperature could be determined by ordering the patient to take some exercise. An elevation of 0.5° to 1.5° in the afternoon, or after some excitement, or exertion, lasting about half an hour may be observed in some persons who have no tuberculosis, as was mentioned above; with the phthisical, however, it is more lasting. It appears that a large proportion of patients with

[1] Jour. Am. Med. Assn., 1919, **62**, 31.
[2] Am. Jour. Physiol., 1918, **46**, 244.

early tuberculosis have a subnormal temperature in the early morning hours, some recording as low as 96° F., before getting out of bed.

When interpreting fever in the early stages of phthisis, we should follow Daremberg's[1] suggestion and consider the difference between the highest and lowest temperature. Thus, a patient with a temperature of 99.8° F. at 5 P. M. has not only 1° above normal when his morning temperature is 96.5° F., but 3.3° above normal, and should be considered febrile, and when persisting for some time, it is undoubtedly of tuberculous origin, unless some other cause is found.

Symptoms of Fever.—These afternoon rises can also be distinguished from other rises, and from physiological elevations, by the concomitant symptoms which are met with in most cases of incipient phthisis. In the latter there is an acceleration of the pulse-rate far out of proportion to the slight elevation of temperature. Many also have mild chilly sensations, or even a distinct chill, about an hour before the rise in temperature, when the face is pale, and the extremities feel cold. Later the face becomes flushed, the eyes brighten with characteristic brilliancy, which can often be recognized by the experienced observer, and the patient feels warm or hot, tired, fatigued and disinclined to work, and has some headache. It is noteworthy that, despite all these symptoms, the appetite for the evening meal is not diminished, which is not, as a rule, observed in fever due to other causes. *Anorexia is a constant accompaniment of fever, excepting the fever of early phthisis.* This tolerance of fever by the tuberculous manifests itself also in their aptitude to work during the day, and sleep during the night as if they were well, feeling only somewhat tired or languid, when the thermometer reads 101° F., or more. Finally, during the night more or less sweating may occur, which even in early cases may be so profuse as to drench the patient.

Subjective Fever without Elevation of Temperature.—These symptoms, in varying degrees of severity, are only rarely absent in incipient phthisis, and they are excellent guides in our attempts at excluding rises in temperature due to other causes. In fact, the afternoon languor just mentioned is so characteristic of the toxic state of the tuberculous that we often meet it in some advanced cases—notably, fibroid phthisis—which are afebrile. In such cases we may speak of subjective fever without elevation of temperature, first described by Dettweiler. I have seen it in a few cases of incipient tuberculosis. For this reason we must not rely solely on thermometry while treating tuberculous patients. Conversely, fever without subjective symptoms is occasionally, though very rarely, seen in incipient cases and the prognosis is very good indeed.

Provoked Fever.—The heat center is apparently easily disturbed in phthisis and as a result we have usually a labile, or unstable, temperature. Conditions which in the average normal individual have

[1] Tuberculose Pulmonaire, Paris, 1905, p. 59.

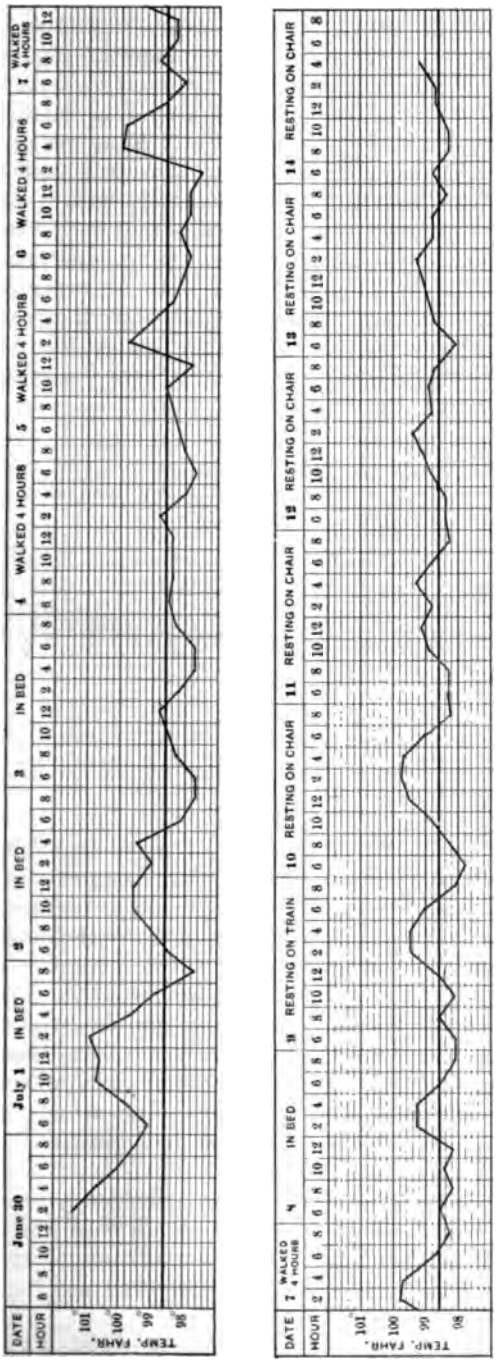

FIG. 36.—Incipient lesion at left apex, temperature taken every two hours, showing the effects of exercise and rest on the temperature. Each time the patient walked a rise in temperature occurred.

no effect on the temperature may elevate it in the consumptive. Thus, a heavy meal, moderate exertion, emotional disturbances like reading or writing a letter, worry, anxiety, and excitement, especially during the early morning hours, may raise the temperature from 1.5° to 3° F. and more. I have seen the excitement of a medical examination raise the temperature of a patient in my office 3.5° within one-half hour, and in European sanatoriums it is a routine measure to inject water at the beginning of a course of tuberculin treatment with a view of ascertaining whether the febrile reaction is really due to the tuberculin or to emotional disturbances. On visiting days in sanatoriums a large proportion of patients have higher fever than on other days. It has also been observed that a change in residence, as the admission into an institution, a railway journey, giving a sanatorium patient leave to spend a day with his family, etc., may elevate the temperature of the consumptive.

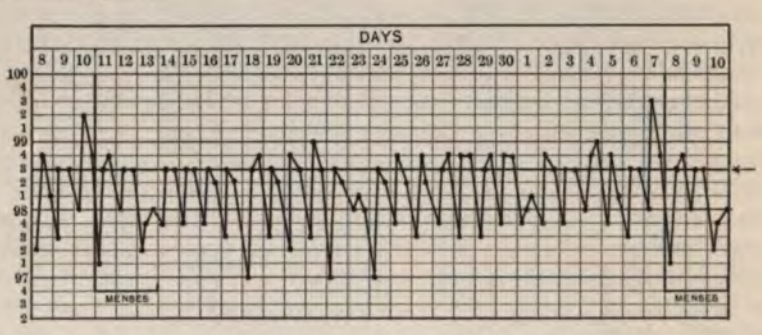

FIG. 37.—Female, aged nineteen years. Premenstrual fever in an afebrile case of incipient tuberculosis. (Bray.)

This *fièvre provoquée*, first described by Daremberg, and then again by Penzoldt,[1] can be utilized for diagnostic purposes in cases suspected of incipient phthisis. When we have a patient presenting indefinite symptoms and signs of tuberculosis, but the temperature is normal, we may take the temperature before and after active exercise, and if it is raised 1° F. or more, we are probably dealing with a case of incipient tuberculosis. The usual rule is to let the patient walk about two miles and note the effect. My way has been to ask the patient to take his rectal temperature before he starts out for my office, and then walk one and a half or two miles while coming. Immediately on his arrival his temperature is again taken, preferably with the same thermometer.

A rise of 1° or more in the temperature after such a test is highly suggestive of tuberculosis. Daremberg insists that it is conclusive. Combined with other symptoms and signs, it is undoubtedly of great value. But in obese persons this may be observed without any tuber-

[1] Handbuch der Therapie, Jena, 1910, **3**, 188.

culous lesions in their lungs and the same is true of anemic, especially chlorotic young women, and in individuals with focal infections in any location—the gums, sinuses, Fallopian tubes, etc. But in physiological rises after exercise the elevated temperature again sinks to normal within half an hour of rest, while in the tuberculous it lasts much longer, two hours, or even more.

Menstrual Fever.—In women the fever may be more accentuated during the menstrual period, which at times is of diagnostic importance (Fig. 37). We must, however, remember that in many non-tuberculous women slight elevations of temperature are observed a few days before or during that period. But in the phthisical we meet not only with elevation of temperature, but occasionally also with an increase in the number of rales over the site of the lesion, hemoptysis, and pleuritic pains. Macht[1] says that "the rise in temperature may occur in afebrile patients, that is, patients who ordinarily run no fever, as well as in those who run a slight temperature throughout the month. These rises may occur in early cases as well as in advanced, and in the former are of considerable diagnostic importance. If a patient shows a constantly recurring menstrual rise in temperature, and pelvic disease cannot be found, a tuberculous process should always be borne in mind."

In most cases the fever declines with the appearance of the flow; it may last several days, or only a few hours. Sabourin[2] has shown that in certain women the menstrual fever lasts three weeks and leaves the patient only one week before the onset of the next menstruation. In these cases it is of grave importance; the patients "are killed by their courses," as Sabourin says.

Many authors, notably Vandervelde, Sabourin, Wiese,[3] C. A. Welch,[4] E. C. Morland,[5] and others, state that premenstrual fever indicates latent or active tuberculosis and should be given attention when attempting to make a diagnosis in doubtful cases. This premenstrual fever occurs a few days before the onset of menstruation and may continue throughout the days of the flow. Considering that it has been found that in from 40 to 50 per cent of tuberculous women there is hyperthermia before and during that period, while in healthy women the percentage is considerably less, these authors maintain that it is of considerable diagnostic value, and that the absence of menstrual fever excludes active tuberculosis.

According to Macht, these rises in temperature, when reaching high, are an evil omen prognostically; on the other hand, if they grow less, or disappear altogether, it is a sign of a cured, or an arrested condition.

Evaluation of Fever in Tuberculosis.—In the usual case of chronic phthisis in the incipient stage there is a subfebrile temperature which

[1] Am. Jour. Med. Sci., 1910, **140**, 835.
[2] Rev. de méd., 1905, **25**, 175.
[3] Beitr. z. Klin. d. Tuberk., 1912, **26**, 335.
[4] Lancet, 1910, **1**, 639.
[5] Ibid., 821.

is often overlooked, unless the thermometer is used every two hours for a week or two. The feeling of languor which overtakes the patient during the afternoon is often taken as an indication of neurasthenia, the anorexia is attributed to dyspepsia, and the real cause overlooked. From Fig. 38 it will be seen that if in this case the temperature had been taken only at 8 A.M., 12 M., and 8 P.M., as is usually done, the febrile reaction at three to six would have been overlooked, and the patient pronounced afebrile. In rare cases, these febrile reactions occur during the night and thus escape detection. Still rarer is the so-called "reversed type" of fever, the febrile reaction occurring during the early morning hours. It appears that the prognosis is unfavorable in the last class of cases.

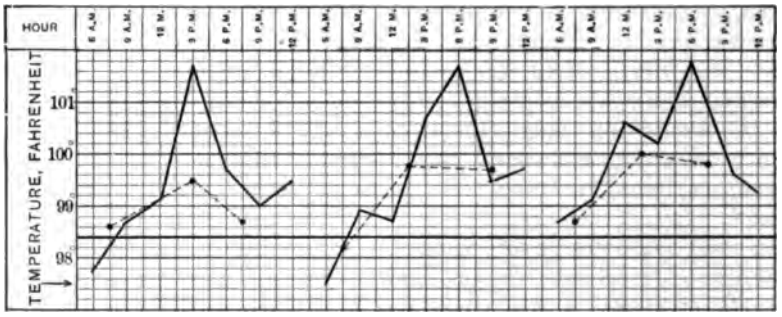

FIG. 38.—Incipient phthisis, active lesions in left apex. Temperature taken every three hours (black line) shows daily exacerbations of the fever reaching 102° F. in the afternoon. This exacerbation would be missed if temperature was only taken three times a day, at 8 A.M., 12 M., and 7 P.M., as is shown by dotted curve.

Since a subfebrile temperature for one or two days is no conclusive proof of the existence of active phthisis, because such ephemeral hyperthermia may be due to other causes, and also because there are afebrile days during the incipient stage of phthisis, the temperature should be taken continuously for two or three weeks in doubtful cases before arriving at a conclusion. The readings thus plotted on the chart are the best graphic criteria for diagnosis.

The slight afternoon rises in temperature characteristic of incipient phthisis are not exclusively met with in this disease; there are other conditions which may produce hyperthermia for weeks, greatly simulating phthisis. For this reason we must not hastily decide in favor of this disease unless there are other symptoms and signs of lung disease. I have had under my care a woman who was treated for several months in a sanatorium, then handed over to surgeons for operation for gall-stones, and while convalescing after the operation another diagnosis of tuberculosis was made. The woman was then admitted to the hospital under my care and for three months the afternoon temperature was almost invariably elevated 1° to 3°. We finally gave

her work as a nurse and she worked during the succeeding six months quite hard and has not developed phthisis, nor shown any indications of the disease on physical exploration of the chest. I knew several individuals who had subfebrile temperature almost every afternoon, yet no cause could be found; however, they continue healthy. These afternoon rises in temperature, when not due to tuberculosis, are mainly found in women. Anemia, especially chlorosis, and occasionally pernicious anemia, may be the cause. However, an examination of the blood clears up the case. Purulent conditions of the nose and accessory sinuses, pyorrhea, chronic inflammatory conditions of the tonsils, chronic otitis media, non-tuberculous bronchiectasis, pyelitis, diseases of the female genitalia, cirrhosis of the liver, hyperthyroidism, syphilis, Hodgkin's disease, pernicious anemia, leukemia, malignant neoplasm of the lungs, etc., may be accompanied by subfebrile temperature. These are but a few of the conditions which must be looked for in doubtful cases.

After all, purely hysterical fever must be borne in mind when everything else has been ruled out. There is no question but that it does occur, although our modern views of the pathogenesis of fever are against it. This appears to be one of the many paradoxes in clinical medicine.

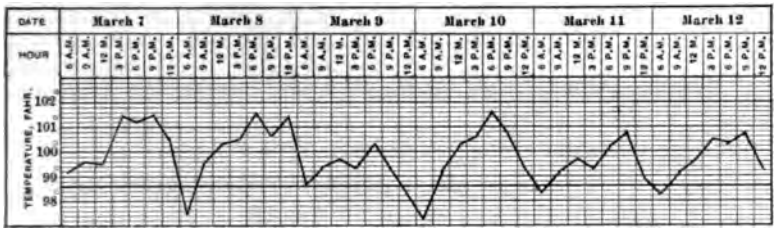

FIG. 39.—Fever in incipient tuberculosis. Temperature taken every three hours.

In evaluating the significance of the temperature range in active phthisis, we may be guided by the rules laid down by Harris and Teale:[1] The higher the day temperature, the more active the disease, except in a few rare instances (the so-called "reverse type") where the ordinary fluctuations are reversed, and the evening temperature remains lowest throughout the whole course of the disease. But whether the normal or the inverted remissions take place, the lowest temperature is always above normal, and so long as it follows this course, it may be assumed that active deposition of tubercle is taking place, even though the physical signs remain for the time unaltered.

Most patients with fever lose in weight, but there are many exceptions, and patients as well as physicians are apt to judge a case more

[1] Treatment of Pulmonary Consumption, London, 1895, p. 314.

by the scale than by the thermometer. This is wrong. There are cases of phthisis, especially those in whom the fastigium occurs during the night, that remain stationary or gain in weight, while the process in the lungs keeps on progressing. In other words, neither fever nor the weight alone should be taken as a criterion for prognosis, but all the concomitant symptoms and signs should be considered in this connection.

On the other hand, the absence of pyrexia, while a good sign in most cases, is not conclusive evidence of the mildness of the process, especially when other symptoms of active disease are present. I have seen many patients in whom the temperature never exceeded 101° F., or was even less, still the anorexia, emaciation, cough, hemoptysis, etc., were all active in bringing them to a fatal termination. This is especially seen in cases which have lasted for some years. The organism has adapted itself to the disease and does not react any more to the same degree that it does usually, and its defensive forces are in abeyance. It may be observed in patients with any lesion, not excluding those with large, but usually dry, cavities in the lungs.

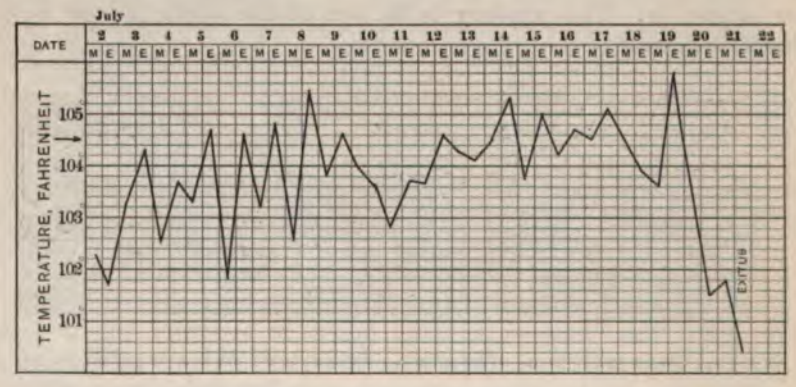
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FIG. 40.—High, continuous fever in the terminal stages of pulmonary tuberculosis.

Types of Fever in Chronic Tuberculosis.—In progressive and also in advanced cases of phthisis the fever is not typical, and a diagnosis cannot be made from an analysis of the temperature curve alone, as is often the case in malaria, relapsing fever, typhoid, pneumonia, etc. In phthisis we may meet with any type of hyperthermia in different patients, and in the same patient at different times, depending on the activity of the process, mixed infection with pyogenic organisms, softening of lung tissue, free drainage of necrotic foci, mobilization of tubercle bacilli into the blood stream, etc. Very frequently we note in the same patient different types of fever at different times, and passing into the other suddenly or gradually. Under the circumstances we cannot speak of a typical tuberculous fever. But we meet with certain

temperature curves which, at times, serve as good and reliable guides in our attempts at ascertaining the condition of the patient, the presence or absence of complications, and especially when attempting to formulate a prognosis.

Continuous Fever.—This is met with especially in cases with extensive pneumonic involvement, in acute pneumonic phthisis, in tuberculous bronchopneumonia in children, and in acute miliary tuberculosis. In chronic phthisis which has pursued a favorable course, when a continuous temperature develops after a pulmonary hemorrhage, or without any visible cause, we may conclude that there has occurred an extension of the process in the lungs; and if this high, continuous temperature—even when it does not exceed 103° F.—lasts more than three or four weeks, the prognosis is very grave and a fatal issue may be looked for. In some cases a slight improvement may occur, but it is noteworthy that they are never cured. Combined with dyspnea, cyanosis, and prostration it is an indication of miliary tuberculosis, which is a frequent terminal phenomenon in chronic phthisis.

Cyclic Fever.—In many cases of chronic phthisis we meet cyclic or undulating types of hyperthermia. The patient is never free from fever, but for two or three days during the week the maximum reading reaches 102.5° or 103.5° F., or even more, while the other four or five days it is much lower—100.5° to 101.5° F. These wave-like fluctuations may appear more or less periodically for months and not only show variations during each week, but the febrile waves may appear at greater intervals, every two or three or four weeks, as can be seen from Fig. 41. It is seen in cases in which old foci are

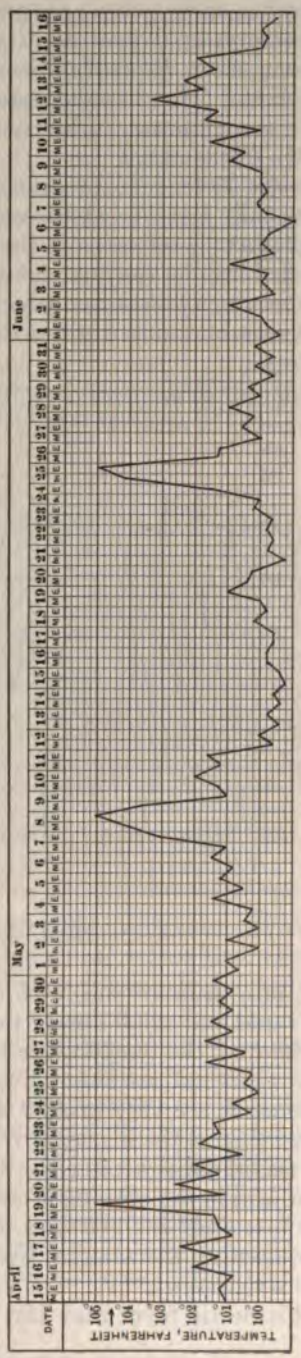
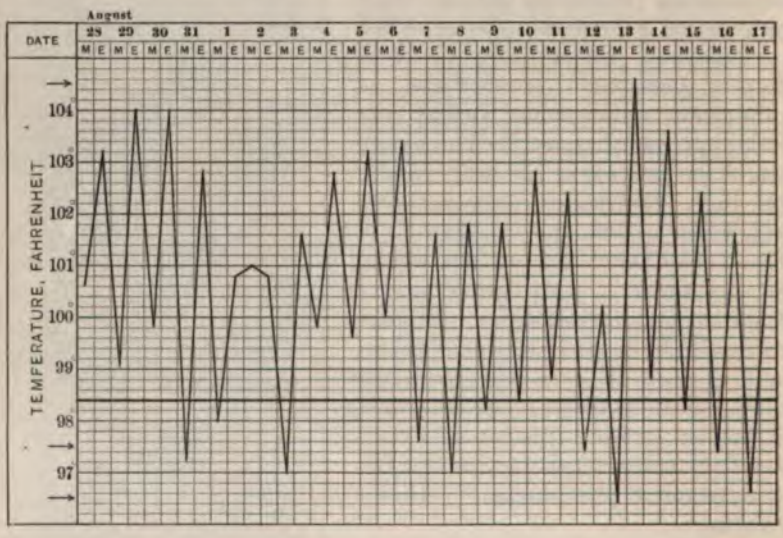


FIG. 41.—Active tuberculosis with continuous fever, marked by acute exacerbations, occurring at irregular intervals.

softening, or the pulmonary process is extending, and each exacerbation of the fever is an expression of a new area of involvement which may, in many cases, be easily discerned by a careful physical exploration of the chest. The diagnostic and prognostic significance of undulating fever in pulmonary tuberculosis has recently been treated in detail by R. Burnand.[1]

Hectic Fever.—In progressive disease these types of hyperthermia are usually followed at the end by hectic fever (Fig. 42). In cases in which there is softening in the lung, the necrotic tissue being gradually expelled leaving cavities, the temperature chart tells the story. There are morning remissions during which the temperature is nearly normal, or even subnormal, while in the afternoon there may be a chilly sensa-


August
DATE
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98
97°

FIG. 42.—Hectic fever in advanced cavitary phthisis.

tion, or a distinct chill, with chattering of the teeth; the pulse, which was rapid and small during the apyrexial morning hours, is even more accelerated, the temperature begins to rise, reaching 103°, and in some cases even 105° at about five in the afternoon. The nightsweats in these cases are very profuse and exhausting.

The time of the highest fever in these hectic cases is variable. Often the maximum is attained in the afternoon, but in many it is around noon, and in the evening it may be normal. If in such cases it is taken only mornings and evenings, we may find a record of normal temperature, because the midday rise, which may have been quite high, has been overlooked.

This hectic fever may last for weeks, or even for months, during

[1] Ann. d. méd., 1919, **6**, 110.

which time the unfortunate patient is reduced to a skeleton by the fever and the accompanying anorexia and diarrhea, which are hardly ever lacking. The frightful appearance of the bundle of bones with hardly any visible muscles, which have atrophied extremely, covered by a clammy, muddy skin; the skin emaciated but edematous around the ankles and knees; the lips and fingers cyanosed, the eyes deeply set in the orbits, the temples sunken, are disheartening to the physician making his rounds in the hospital; he feels helpless when the slowly sinking, but still struggling, human being gazes, appealing for assistance which cannot be given. It is noteworthy that with all this material decay the intelligence, and often the hopes and aspirations of the patient, are well retained, and he begs for the relief of some minor, and comparatively insignificant symptom, such as the cough or diarrhea, saying that if this is removed he will feel in excellent condition; many beg to be sent to some distant clime where they are sure of a cure.

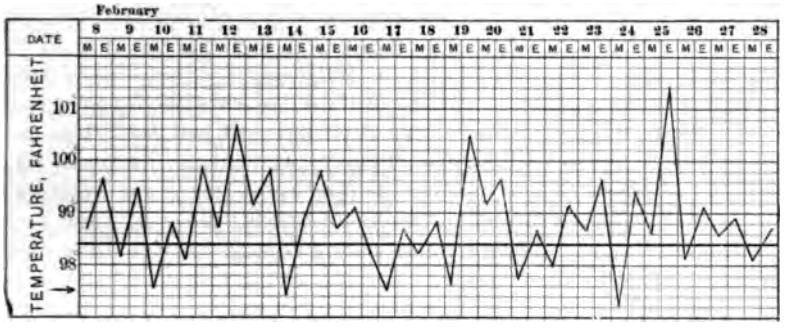


FIG. 43.—Irregular fever in advanced tuberculosis of the lungs with intestinal complications.

At the terminal stages there may be irregular fever; the curve of one day differs from that of the other. Saugman[1] states that this is a good sign of intestinal tuberculosis when occurring in the earlier stages of the disease (Fig. 43).

Subnormal Temperature.—The subnormal temperature seen in many incipient cases during the morning hours has already been mentioned. But we also meet with patients in the advanced stages of the disease who present subnormal temperature throughout the day and night for weeks; the mercury never rises above 98.5° F., and early in the day it may be as low as 96° or 97° F. The disease may be active and even progressive, yet the thermometer gives no indication of it. I have had many of these cases in my hospital service. I find it is usually an indication of excavation, just as fever is an indication of infiltration, caseation, and softening of lung tissues.

[1] In Brauer, Schröder and Blumenfeld's Handbuch der Tuberkulose, 2, 284.

Subnormal temperature is also seen in fibroid phthisis, and in emphysema complicated by tuberculosis, in both of which the disease runs a chronic, sluggish course. Many keep disabled for years, though not confined to bed, but they never fully recover. A subnormal temperature is also seen on rare occasions in a subacute case of phthisis, which suddenly takes a turn for the better after the necrotic tissue in the lung has been eliminated from that organ and a cavity remains. In this class recovery may take place, as I have seen in several instances.

A sudden drop in the temperature, combined with dyspnea and cyanosis, in a febrile case of phthisis may mean a spontaneous pneumothorax, or a rapid extension of the necrotic process in the lung overwhelming the patient or, rarely, complicating and terminal miliary tuberculosis. The prognosis in either event is grave indeed. In many extremely emaciated consumptives the temperature is at times subnormal for several days before death and this does not exclude miliary tuberculosis.

Apyretic Tuberculosis.—In old chronic cases of phthisis we may have a normal temperature for months, though the process in the lungs keeps up. This is seen in fibroid phthisis, in phthisis in the aged, and in tuberculous pleurisy. Many of these patients live for years and do not lose in weight. I have seen such patients last for fifteen and twenty years, always ailing, coughing, expectorating, at times having spells of more or less profuse hemoptysis. They are important sources of the dissemination of tubercle bacilli; more so than most of the stormy cases. They are not strong enough for muscular work, but may be moderately efficient at any occupation which does not require undue exertion. We meet these cases mainly among the well-to-do, who can afford to lead an idle life, or among the very poor who have intrenched themselves in hospitals for chronic and "incurable" cases of tuberculosis and, for one reason or another, like institutional life, and stick to it for long periods. We also meet these active, but apyretic, cases among the more cultured classes, who either know how to take care of themselves or, being professional persons, they pursue their vocations, which do not involve muscular exertion, with more or less efficiency. Some are very brilliant, and the type of consumptive drawn by so many writers of fiction is usually copied after the model of this class of patients. It is noteworthy that while most of them are more or less emaciated, we now and then meet one who is actually fat, and may even be placed in the category of the obese. They usually suffer from dyspnea, because of the fatty heart and pulmonary fibrosis.

Phthisis in the aged also runs an apyretic course at times and, because they do not cough excessively, the disease may not be recognized.

It appears that there are great differences in the reactive powers of different persons suffering from phthisis. In some the fact that they have normal temperature is no proof that the disease is benign, especially if other symptoms of active disease are present. I have seen patients whose temperatures hardly ever exceeded 101° F., yet

they wasted, perspired, and had exhausting diarrhea; they finally died with a low temperature. While the temperature curve is an excellent guide as to the tendencies and progress of the disease, these apyretic cases must be judged more by the general symptoms and the physical signs than by the thermometrical findings, as has already been shown.

Fever Due to Complications.—During the course of phthisis fluctuations in the temperature usually go hand-in-hand with the activity of the disease, and each elevation or depression in the temperature curve may be explained by the findings in the chest through physical exploration. But there are exceptions. Many elevations of the temperature are due to non-tuberculous complications. Thus, as will be seen from Fig. 44, malaria may complicate phthisis and create confusion, unless the blood is examined and the malarial parasite is found.

Other complications to be mentioned are constipation, acute gastritis, tonsillitis, influenza, pleural effusions, etc. These may be the cause of a sudden elevation of temperature in a case in which the tuberculous process is proceeding rather favorably. Careful examination usually reveals the cause of the pyrexia.

A rise in the temperature in a tuberculous patient may be due to the administration of certain drugs, mostly of the sedative and hypnotic class, as has been pointed out by Sabourin[1] and Mantoux.[2] The writer has repeatedly observed that after the administration of opium, or its derivatives, morphine, codeine, heroine, dionine, etc., or chloral, veronal, sulfonal, trional, etc., there is a rise in the temperature during the succeeding twenty-four hours. A rise of this kind is especially vivid when occurring in an afebrile patient to whom one of these drugs has been administered. The fever lasts no more than twenty-four hours, as a rule, but I have seen cases in which it lasted longer. Hypodermic medication is more apt to act this way, and injections of salt solution may also elevate the temperature.

Diagnostic and Prognostic Significance of Fever in Phthisis.—Summarizing the results obtained in this section, we may say that *in a patient who shows a distinct elevation of temperature during the afternoon for several weeks, and no other cause can be found, tuberculosis is to be thought of.* If it is provoked by moderate exercise, and persists after more than an hour of rest, it is almost pathognomonic of phthisis. If with it there are other symptoms, such as nightsweats, languor, loss of weight, cough, emaciation, etc., tuberculosis is in all probability the cause, even if the physical signs are not definite. The diagnosis is more certain if the morning temperature is subnormal.

Instability of the temperature and pulse is found in nearly every case. But it is not pathognomonic of tuberculosis. Overexertion will raise the temperature in every individual with a focal infection of any

[1] Rev. gén. de clin. et de thérap., 1906, **20**, 639.
[2] Rev. de la tuberc., 1907, **4**, 395.

kind in the body. Moreover, any aberration of the sympathetic or endocrine system may be accompanied by instability of the pulse and temperature. This is especailly true in cases of hyperthyroidism. The fact that this is often accompanied by other general symptoms simulating tuberculosis—tachycardia, emaciation, cough, sweating, malaise, fatigue, etc.—makes it more difficult at times to differentiate the two conditions. The effects of nervous tension, anxiety and suspense have been spoken of above.

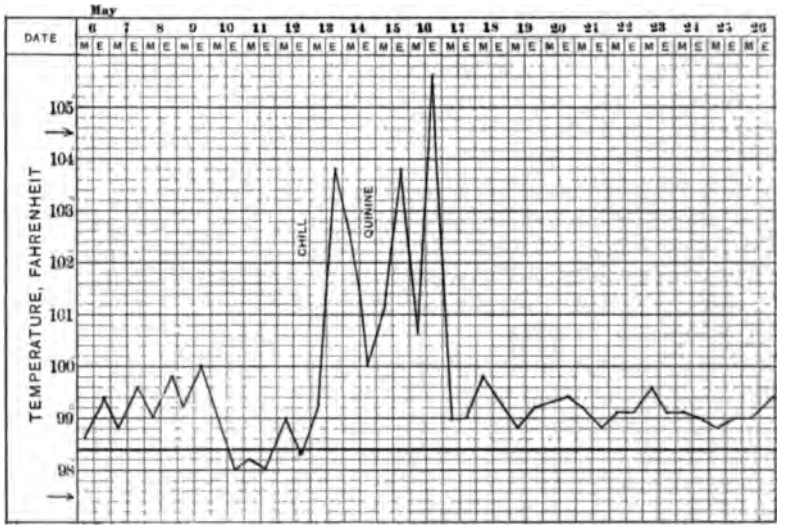


FIG. 44.—Malaria complicating phthisis.

In the course of the disease a *high temperature during the day, never touching the normal, and ascending in the evening is an indication of progressive activity of the process in the lung.* The disease is progressing slowly, or is even quiescent, when the temperature in the early morning on rising is subnormal or normal and remains so during the day, not rising above 101° F. late in the afternoon or evening.

High, continuous temperature, above 103° *F., is an indication of extension or dissemination of the disease in the lung,* and if it lasts for more than a month, a fatal issue is to be expected; even if some improvement is noted, recovery should not be expected.

Hectic fever, with normal or subnormal temperature in the morning, and high fever, 103° *or more at midday or later, is an ill omen.* While the patient may keep on in this condition for weeks or months, he will in all probability never leave his bed alive.

In most cases, *absence of fever is an indication of an improvement or a cure of the disease, but there are many exceptions,* and the other constitutional symptoms must be considered when formulating a prognosis.

A subnormal temperature, when coming on suddenly, is a bad sign. When chronic, lasting for several weeks, however, it is not incompatible with an inefficient, though not necessarily an inactive, life.

NIGHTSWEATS.

Nightsweats have at all times been considered pathognomonic of phthisis. A prolonged cough will not alarm the average person, but when it is associated with nightsweats, he will soon consult a physician with a view of ascertaining whether or not he is tuberculous. They are met with quite early in the disease in many cases; at times when the characteristic symptoms and physical signs are lacking, but in advanced cases their severity does not depend altogether on the extent of the lesion.

Causes.—The causes of nightsweats are obscure. Traube attributed this phenomenon to the compensatory activity of the skin when the pulmonary respiratory area is diminished, but we meet them in cases with but little damage to the lung. Gustav Heim[1] is of the opinion that the products of cell disintegration, and especially the toxins produced by the bacilli, stimulate the sweat center directly or reflexly, just as after childbirth the remains of the placenta may produce sweating. It is an attempt on the part of the body to rid itself of harmful matter, as it is excreting carbon dioxide in the sweat when this is excessive in the blood. Smith and Brehmer have attributed the nightsweats to the quick change of the tachycardia of the day to the bradycardia of the night. It is more rational to see in nightsweats a result of the decline of the high temperature in the afternoon and evening to the low temperature of the remission during the early morning hours. This phenomenon is seen during, and soon after, the crisis in many other febrile diseases. The diminution in the pulse-rate and in the blood-pressure, owing to the atony of the bloodvessels, especially those of the skin, favors sweating and assists in ridding the body of the toxins.

Cornet looks upon nightsweats as due to the absorption of the proteins of the tubercle bacilli and other microörganisms secondarily implanted in phthisical lesions. The toxins are absorbed into the blood stream and they stimulate the heat center, thus causing fever; and also act upon the sweat center in the cord and medulla and the peripheral secretory glands and thus produce perspiration. He shows that this also confirms the fact that, in spite of the great disturbance, the diminished excretion of fluid, and the greater difficulty in the elimination of carbon dioxide which is characteristic of the chronic course of the disease as compared with acute phthisis, the secretion of sweat is incomparably less in the former, owing solely to the more gradual absorption of the toxins.

[1] Ztschr. f. Tuberk., 1910, **16**, 365.

Symptomatology.—Nightsweats usually occur in the second part of the night, about 2 to 4 A.M., in typical cases. The patient retires with some fever, and in hectic cases may have had a chill on the preceding afternoon, sleeps rather restlessly, is disturbed by dreams or by cough, and wakes up during the early morning hours drenched with perspiration. At times, changing the night- and bedclothes may prevent their recurrence during the same night, but in many cases this is of no avail, as the sweats again trouble the unfortunate victim.

In the milder forms, the sweating may be local, on the forehead, the neck, the chest, etc. Rarely it is noted on only one side of the body, usually the one corresponding to the pulmonary lesion.

In the progressive and hectic cases the sweating may be so profuse and drenching as to exhaust the patient, who often begs for the relief of this symptom alone which, together with the diarrhea, is instrumental in relieving him permanently from his earthly sufferings.

It is important to mention that the nightsweats do not directly harm the patient, considering that only 1 per cent of solids is eliminated in this way, of which 0.7 per cent is salts, mainly uric acid. Only so far as disturbing sleep is concerned are nightsweats harmful. In children their diagnostic significance is less than in adults. (See Chapter XXIV.)

In some cases the disease runs its course without any, or only with slight nightsweats. Kuthy found that 37 per cent of his patients had nightsweats during the first stage of the disease. In the third stage, 61.5 per cent. According to this author, women are more apt to sweat profusely than men. But Louis found only 10 per cent of cases without nightsweats, and at the Phipps Institute they were absent in 41 per cent of 3344 cases.

In the evolution of phthisis it is observed that, as a rule, the sweats run hand-in-hand with the fever and the general condition of the patient. During afebrile periods they are absent to return with an acute exacerbation. There are said to have been observed cases of nightsweats without fever, but my experience leads me to believe that the fever was overlooked in such cases. *One of the best signs of improvement is the complete disappearance of the nightsweats.*

Nightsweats may be prevented in a large proportion of cases by the adoption of hygienic bedding and coverings during sleep, as will be shown in another part of this book.

Sweating appears to be easily provoked in the phthisical. Kuthy and Wolff-Eisner say that not only consumptives, but also those "predisposed" sweat easily, who, when waking, find themselves bathed more or less in perspiration. Mild exertion, grief, worry, excitement, etc., may be followed by more or less profuse perspiration, general or local. In a large proportion of patients we see sweating in the armpits during medical examination, even in patients who do not sweat during the night. We also meet with patients who sweat during the day while taking a nap, etc.

While most authors, notably Cornet, state that the sweat does not carry infection, investigations by Piéry have shown that it may contain bacilli which are pathogenic to animals. However, this has not been confirmed by other investigators. Probably the sweat collected by Piéry was contaminated by bacilli after it was secreted. Salters showed that hypodermic injections of the sweat into animals act like tuberculin, which may be expected, considering that it assists in eliminating tuberculous toxins from the body.

CHAPTER X.

HEMOPTYSIS.

Frequency.—To the layman the most reliable symptoms of pulmonary tuberculosis is blood-spitting and many physicians share this view, although we know that a large proportion of cases of phthisis pursue their course and terminate in recovery, or fatally, without any hemoptysis, while in many patients hemoptysis is not due to tuberculosis. The statistics of the frequency of this symptom vary considerably, some finding it in but 25 per cent, while others report as many as 80 per cent having had hemorrhages during the course of phthisis. Sokolowski says that advanced consumptives who did not bleed from time to time are only rarely met with. Louis found this symptom in 65 per cent of cases; Walshe[1] in 80 per cent; Wilson Fox[2] says that more than one-half of all cases of phthisis present this symptom in some part of their course; Williams found it in 70 per cent; Sorgo[3] in 38 per cent; Condie in only 24 per cent; Elmer H. Funk among 373 patients with advanced disease, in 44 per cent; among 167 patients traced to the end in 54 per cent, and at the Phipps Institute at Philadelphia, it was found in 49.9 per cent of 4466 tuberculous patients.

These wide differences in the percentages are easily explained by the fact that the authors have not taken their figures from comparable material. Some have spoken only of fatal cases, others of cases in their private practice, while still others have taken hospital records as their criteria. In the latter classes the patients were observed only for a short time, and hemorrhages which may have taken place later have not been considered.

Anders[4] found in a series of 5302 cases that 36.6 per cent had hemoptysis. He emphasizes, however, that not all were followed until the death or recovery of the patients, but many were discharged during the course of the affection. In fact, among 289 cases in private practice, kept under observation for a longer time, as a rule, hemoptysis occurred in 41.8 per cent, but it is to be recollected that even these patients were under observation for less than half of their duration. Hemorrhage is more apt to occur in advanced cases, and those who base their calculations on early cases in sanatoriums are likely to find low percentages, while when only fatal cases are taken the percentages will be too high.

[1] British and Foreign Med. Chir. Review, 1849.
[2] Diseases of the Lungs and Pleura, London, 1891, p. 785.
[3] In Brauer, Schröder and Blumenfeld's Handbuch d. Tuberkulose, **2**, 250.
[4] Jour. Am. Med. Assn., 1907, **49**, 1067; 1909, **53**, 455.

Pathology.—The diagnostic and prognostic significance of hemoptysis can only be appreciated when we have a clear understanding of the anatomical changes responsible for the bleeding. There are several varieties of pulmonary lesions which may bring about extravasation of blood from the lung tissues: Local inflammatory or active hyperemia; ulceration of a bloodvessel, and aneurysmal dilatation of bloodvessels are the most important in phthisis.

The initial hemoptyses are said to be caused merely by localized, active inflammatory hyperemia. In other words, they are of the same origin as the rusty sputum of pneumonia and the hemorrhages in influenzal bronchopneumonia. This bleeding, caused by diapedesis and active congestion of the pulmonary parenchyma, may be profuse, though in most cases only blood-streaked sputum is thus brought out. On the other hand, blood-streaked sputum does not invariably mean that it is caused by localized hyperemia and that the lesion is not serious, because not all the extravasated blood is brought out through the mouth. Quite some of it remains in the lungs and bronchi, and is more or less quickly absorbed, as was shown by Nothnagel. *When the hemorrhage is not profuse we must not conclude that the case is mild, or that the lesion is not extensive.*

When the pulmonary lesion proceeds from infiltration to caseation, then to softening, and finally to liquefaction, it undoubtedly implicates the bloodvessels that pass through it and produces in them the same changes as in the lung tissue. It is therefore strange at first sight that, considering the ulcerative processes and the destruction of tissue, hemorrhages do not occur more often. But this is explained by the strong tendency to the formation of thrombi in the bloodvessels, excepting in very acute cases. In chronic cases there usually occurs a narrowing, or complete obliteration, of the veessl by the growing tubercles which, when finally ulcerating, may leave an erosion through which the blood can flow more or less freely until it is occluded by a thrombus. Moreover, the increased blood-pressure at the infected and inflamed area dilates the softened vessels, and causes small aneurysms, the *aneurysms of Rasmussen*, which have been described elsewhere (see p. 176). This is clear when we bear in mind that the bloodvessels in the lungs are terminal branches of the pulmonary artery.

Most cases of hempotysis end in recovery, and the pathological changes in the lung at the time of the bleeding can only be surmised, but in fatal hemorrhages we often have an opportunity to observe the anatomical changes. Here we usually find that the source of the bleeding was an exposed vessel, left bare after the surrounding pulmonary tissue had softened and was eliminated. The loss of support, as well as the pathological changes in the perivascular tissues, and the erosions of the tunicæ adventitia and media, lead to aneurysmal dilatations of the inner coat which give way to the pressure exerted on them by the circulating blood.

The rupture of these aneurysms at times strikes down a patient

who is on the road to recovery when a hemorrhage occurs like a storm out of a clear sky. When the cavity into which the aneurysm, or the lacerated artery, opens is small, the extravasated blood usually coagulates, and the clot obstructs the opening of the bloodvessel, thus stopping the bleeding. But in large cavities, or when the blood is deficient in coagulability, which is not rare, the bleeding keeps on until the patient dies of acute anemia. I have seen at autopsy a large cavity filled with about a quart of blood which killed a patient during the night. After clearing out the clots we found an eroded artery about 2 mm. in diameter, and passing a probe through it, we found it only about 6 cm. from the pulmonary artery. This patient had such a sharp hemorrhage that he was unable to call for assistance.

In more acute cases of phthisis, in which the destruction of lung tissue is going on at a rapid pace, the hemorrhages usually come from ulcerating erosions of large pulmonary vessels and may prove fatal immediately. Here there is no time for narrowing of the bloodvessels, thus preparing it that in case of rupture it may be easily repaired by occlusion with a thrombus which saves the majority of chronic consumptives from death due to this cause. In acute pneumonic phthisis, which very often begins with sharp and profuse hemorrhage, I have usually been able to find signs of cavitation when the acute process subsided and the disease pursued a chronic or subacute course. This confirms the view that profuse hemorrhage is not caused by mere active inflammatory hyperemia, but by actual erosion of a bloodvessel.

In fibroid phthisis the sources of hemorrhages are lacerated, dilated or varicose bloodvessels which pass through bronchiectatic cavities, characteristic of this form of the disease, and also oozing from capillaries or arteries which traverse the granulations on the walls of the cavities. The bleeding is therefore not profuse, as a rule, but it is recurring in many cases.

Effects of Hemorrhage.—It has been found experimentally that animals can withstand the loss of one-half of the total amount of blood in the body. When death does ensue, it is not due primarily to the diminution in the number of red blood cells, but because of the sudden drop in the blood-pressure resulting from scanty filling of the bloodvessels and the heart. At first the bloodvessels contract and thus maintain the blood-pressure, but later they dilate and a proportional hypotension results. When the hemorrhage is not extremely copious and rapid, the bloodvessels are soon filled with fluid absorbed from the body juices. Therapeutically we attain the same end by transfusion. The result is that in acute primary anemia the pulse is rapid owing to the rapid heart action, but it is small owing to the oligemia. With excessive loss of blood cerebral anemia ensues. Fainting due to this cause has been observed when 500 cc of blood is rapidly lost; 1500 to 2000 cc of blood rapidly lost from the body is fatal. In anemic tuberculous patients, who have an oligemia even before the intercurrent pulmonary hemorrhage, even a smaller amount may end fatally.

Initial Hemoptysis.—Of great interest is hemoptysis as an initial symptom of phthisis. But statistics on this subject are also at variance, because we meet with many patients who have been coughing and presented other symptoms of tuberculosis for months, or even years and paid little attention to them until a hemorrhage brought them to their senses. Here it would not be correct to consider the hemoptysis as the first symptom.

In a study of 1932 cases Reiche[1] found that 9.2 per cent had more or less profuse hemorrhage at the beginning of the disease, and in one-fourth of these it was rather copious. He finds that those who bleed at the beginning are more apt to bleed during the course of the disease than those who do not; the ratio is 57.9 per cent and 31.7 per cent. Sorgo found during a period of observation extending over ten years that 12.9 per cent of 5872 patients had initial hemorrhages. Kuthy[2] reports that while 54.3 per cent of his patients had hemoptysis, only two-fifths of these (22.3 per cent) were initial hemorrhages. Anders arrives at the conclusion that in about 10 per cent of cases of phthisis, hemoptysis first directed attention to, and is almost invariably followed by, demonstrable and conclusive evidence of the disease; but in not less than 25 per cent of all cases of chronic pulmonary tuberculosis, hemoptysis is among the ushering-in symptoms of the active recognizable period of the affection.

Hemoptysis at the Onset of Phthisis—As the first symptom to draw the attention of the patient to his affection, hemoptysis occurs in two different types. We meet it in patients who have felt perfectly well until the instant the hemorrhage made its appearance without any premonitory symptoms. Even close questioning does not elicit any symptoms preceding the bleeding. While at work, or engaged in an animated conversation, or even waking up from sleep during the night, the patient feels a sensation of warmth in the throat, coughs, and expectorates a mouthful of blood; or during a fit of coughing he brings up some blood-streaked sputum. A careful examination of the chest and roentgenography may fail to disclose anything conclusive of pulmonary disease. The temperature is, and remains, normal, the appetite is good, but for a few hours or days the patient continues to bring up dark clots, and when this ceases he is apparently none the worse for his experience. Many of these patients subsequently pass through life without experiencing anything that may lead to the suspicion of tuberculosis. This is seen in those who have passed through an attack of abortive tuberculosis, details of which are given later on. Some patients give a history of such a hemorrhage many years before the onset of active phthisis.

In others the initial hemorrhage continues for several days, and when it finally ceases the patient shows symptoms of phthisis—cough, expectoration, tachycardia, nightsweats, etc. Physical exploration

[1] Ztschr. f. Tuberk., 1902, **3**, 223.
[2] Die Prognosenstellung bei der Lungentuberkulose, p. 299.

of the chest reveals distinct signs of a lesion in one or both apices, and tubercle bacilli may be found in the sputum. The subsequent course of the disease is that of chronic phthisis, though a large proportion of cases are aborted within a few months, and I have met with patients who have had several attacks of hemoptysis at long intervals have shown some indefinite, or even conclusive apical signs, and rarely tubercle bacilli in the sputum, yet they remained well indefinitely.

A different clinical type of hemoptysis is seen in patients who maintain that they had felt quite well, but close questioning reveals the fact that they have been coughing for months, bringing up mucopurulent sputum; that the appetite has failed, and that they have lost weight and strength. In women we may find that they have missed one or more of their periods. They, however, considered these symptoms trifling, and continued at their work; or, consulting a physician, they were told that it was only a slight "cold."

The hemorrhage in these cases is apt to be profuse, and to last for several days because, while insidious in its arrival, the tuberculous process in the lungs has usually progressed quite far; indeed I have met with signs of pulmonary excavations in such "initial" hemorrhages. In the majority of cases physical exploration of the chest reveals a lesion of moderate extent, though on rare occasions we find nothing definite, even with the aid of roentgenography. But the cough, fever, night-sweats, expectoration, etc., continue, and the diagnosis is made without conclusive physical signs. In most cases tubercle bacilli are found in the sputum. It is the slow and prolonged convalescence after the attack of hemoptysis that distinguishes these cases from the initial hemorrhages of abortive tuberculosis.

Hemorrhages during the Advanced Stages.—In confirmed chronic cases of phthisis we may meet with hemoptysis at any period of the disease, though it may be added that it is most frequent in the early and very late stages. The bleeding may be of various degrees, from that of sputum tinged with blood, to the expectoration of several mouthfuls of pure, bright red blood, to a copious hemorrhage during which several pints are brought up within twenty-four hours, and in rare cases it has been reported that as much as three quarts of blood were brought up.

The blood is bright red, frothy, usually mixed with sputum. When bleeding is very profuse the blood may be "blue," or venous. It is evident that in most cases the blood does not coagulate quickly—some clots are seen, but the bulk remains fluid; even the addition of calcium salts, serum and tissue extracts does not enhance its coagulability. E. Magnus Alsleben[1] has added normal blood without increasing its power of coagulation. The reasons for this delayed coagulability are not clear.

Many patients have some premonitory warning before the onset of

[1] Ztschr. f. klin. Med., 1914, **81**, 9.

hemoptysis, and I have had one who could foretell bleeding twenty-four hours in advance. At times there is a rise in temperature, and pains in the chest are aggravated, or the cough becomes more annoying. But in most patients the onset is sudden and unexpected. The patient has a sensation of gurgling or tightness in the chest, followed by a fit of coughing productive of bright red, frothy blood which has a salty taste, and partly coagulates in the vessel into which it is deposited, forming flattened lumps. When very profuse, which is comparatively rare, the patient is overwhelmed and can hardly cough—the blood gushes in an almost steady stream through the mouth and at times through the nose.

The general appearance of the average patient is that of shock—he is prostrated, often out of proportion to the amount of blood lost; his countenance is that of a frightened individual, unnerved, anxious and terrified; the face pale, the extremities cold and clammy. The temperature, which may have been above normal before the onset of the bleeding, suddenly sinks, often to a subnormal degree; the pulse is rapid, soft, and small.

That these symptoms of collapse are not due wholly to the loss of blood is evident from the fact that the family is also panic-stricken, and some are in the same state of collapse as the patient, showing the profound influence this symptom has on the average person.

After getting some reassuring encouragement from his physician, there is usually observed a reaction in the patient—the pulse improves, the face becomes flushed, and the temperature rises to the same degree as it was before the onset of the bleeding, or higher. In many cases there is soon a relapse, the bleeding is repeated within a few hours or the next day, and it may keep on at irregular intervals for a week or more. When it finally stops the patient continues to expectorate dark blood-clots with his sputum for several days. In some cases the bleeding continues for weeks, letting up for a day or two, to reappear; rarely until the patient expires from exsanguination, cerebral anemia, and cardiac asthenia.

In cases with large pulmonary cavities the bleeding may be very copious. The quantity of blood brought out is not all that has escaped from the bleeding vessel. A considerable part is swallowed automatically, and some remains in the cavities or the bronchi, and is subsequently absorbed. The outcome of the bleeding depends on the size of the bloodvessel which has eroded, and the coagulability of the blood. In rare cases the weak and emaciated patient is overwhelmed by the bleeding and is unable to expel it from the lungs, expiring in a few minutes, drowned, or suffocated by his own blood. Other patients make a vain fight for hours, or days, but finally succumb to exsanguination. But the chances of recovery of a bleeding patient with a cavity in the lung are, on the whole, not bad. *An immediate fatal issue is, after all, exceptional; less than 2 per cent of bleeding consumptives die from hemorrhage directly.* The vast majority of hemorrhages are well borne, the patient dying, if at all, from other symptoms or complications.

On the other hand, we meet with tuberculous patients who have made an excellent recovery, but suddenly profuse hemorrhages occur which carry them off within a few hours or days. I was once called to attend a patient who was discharged from a sanatorium three days previously as an arrested case of phthisis. He succumbed to the bleeding. These hemorrhages are fortunately rare, and are usually due to the rupture of an aneurysm in a "dry" and contracted cavity. They can neither be foreseen nor prevented.

Hemorrhages in Fibroid Phthisis.—In this form of phthisis hemoptysis is very frequent. In most cases it is very slight, only blood-tinged sputum being brought up. The patients may feel quite well in general, excepting for the dyspnea and the cough to which they have adapted themselves. But when blood make its appearance in the sputum they are alarmed. I have, however, had some patients who did not mind the blood-tinged sputum much, knowing from experience that it is not at all dangerous. Profuse and even fatal hemorrhages may, however, occur in fibroid phthisis.

Hemorrhagic Phthisis.—There is a form of phthisis which is characterized by frequent and recurrent hemorrhages, the *hemorrhagic phthisis* of the old writers. The bleeding occurs at irregular intervals for years without harming the patient very much. In these patients we may not find any definite physical signs in the chest, no fever, no pronounced emaciation, and but little cough. Only the hemoptysis and, at times, the bacilli in the sputum reveal the condition. I have had under my care at the Montefiore Hospital a woman in whom neither any of the other physicians, nor myself, was quick in making a diagnosis of tuberculosis from the indefinite physical signs and the roentgenogram of the chest. In fact, we had suspected malingering and employed strong measures to make sure that the temperature readings were not influenced by manipulations of the thermometer, and that the sputum was expectorated by the patient, suspecting that there was some deception on the part of the patient, who liked to remain in the hospital. Even during the more or less copious attacks of hemorrhage, which recurred at frequent, but irregular, intervals and often lasted for several weeks, no conclusive physical signs could be elicited in the chest. I have another patient who has bled at least twice a year for the past fifteen years and feels quite well. Andral mentions a patient who bled off and on for sixty years and finally succumbed at the age of eighty to some disease of the chest. These cases are uncommon but we meet them now and then. In some, we find signs of more or less extensive pulmonary lesions which remain stationary, or quiescent, in spite of the recurring hemorrhages. The lesion is benign notwithstanding the tubercle bacilli which are found in the sputum, and at times, though rarely, there may be one hemorrhage which proves fatal. It has been stated that in most of these cases the lesion is localized in the tracheobronchial glands, but it is doubtful whether this pathology explains the recurring hemorrhages.

Exciting Causes of Hemoptysis.—We have seen that while hemoptysis is rather common among consumptives, still many pass through the disease until the end, recovery or death, without this accident. There appears to be some evidence showing that tall persons are more likely to bleed than those of shorter stature, and Wolff states that for this reason women show a lesser proportion of bleeders than men. Strandgaard[1] suggests that the tall patients are more likely to bleed because they have larger hearts and higher blood-pressure, but this view has not been confirmed. While hemoptysis has been seen at all ages, even in infants, still most of the cases occur between fifteen and fifty, probably because at this period most of the cases of phthisis are active.

From Anders's statistics it appears that males are more liable to hemoptysis than females, and prior to the twentieth year of age there is a slight preponderance in favor of the female sex. In Thompson's[2] collective investigation the women showed greater liability than the men. But Anders shows that this increased incidence in the female sex is confined principally to the first two decades of life. After the thirtieth year the number of males preponderates. Females are also less liable to suffer from copious and fatal hemorrhages. My own experience coincides with that of Anders, that *an immediately fatal hemorrhage is relatively rare in women. Initial hemoptysis is also less frequent in women than in men.* Reiche's statistics show that it occurred in 11 per cent of the latter as against only 5.5 per cent in the former; Sorgo found the ratio as 11 and 13.5 per cent respectively; while Berthold Müller[3] found it in equal proportion in both sexes.

Some nineteen hundred years ago Aretaeus described the "hemoptysical constitution" as distinguished by brilliant whiteness of the skin, bright redness of the cheeks, narrowness of the chest, alar scapulæ, slenderness of the limbs and trunk, combined with a certain degree of adipose and lymphatic stoutness. Laennec said that phthisical subjects possessing this bodily configuration are more subject to hemoptysis than others.

Patients with a nervous and excitable temperament are more apt to suffer from this complication than the indolent and phlegmatic. During some animated conversation, overexertion, singing, running, mountain climbing, straining at stool, or as a result of traumatism, hemorrhage may be provoked. But *we should not overestimate overexertion as a factor in the causation of hemoptysis.* Streaky sputum, or mild hemorrhages may be caused by overwork or excitement. But copious hemorrhages are due to rupture of an aneurysm of Rasmussen, or the erosion of a comparatively large branch of the pulmonary artery by a tubercle. Perhaps the fact that the majority of copious and fatal hemorrhages occur during the night shows clearly that overexertion

[1] Ztschr. f. Tuberk., 1908, **12**, 209.
[2] Causes and Results of Pulmonary Hemorrhages, London, 1879.
[3] Ztschr. f. Tuberk., 1910, **13**, 133.

is not the main factor. We are in the dark as to why these hemorrhages are more likely to occur during the night. Consumptives who have been urged on to eat excessively, becoming plethoric, ruddy, and fat, bleed more often than those who eat well, but moderately. Exposure to the inclemencies of the weather may excite hemoptysis, probably by causing an acute localized congestive and pneumonic process at the site of the tuberculous lesion. Coitus may excite it, and I have known two cases of fatal hemorrhage which occurred soon after intercourse.

Certain drugs used extensively in phthisiotherapy, as arsenic, cresote and its derivatives, the iodides, aspirin, etc., are often instrumental in bringing on hemoptysis. It has been stated that residence in high altitudes favors hemoptysis, but it has not been proved; as will be shown elsewhere, the prognosis of hemorrhage appears to be worse in these regions than at sea level.

Some authors have found that there are seasonal influences in the production of hemoptysis, saying that the spring and summer months give the highest incidence, while Anders's collective investigations show that it is most prevalent in the months of December, January, and February; August, September, May, and March, in the order named, seemed to rank next. The experience at the Phipps Institute coincides with that of Anders. Burns[1] says that "barometer changes seem to have little effect on the symptomatology. In a few instances hemorrhages have occurred following a fall in the barometer, but in insufficient number of cases to justify constant relation. It is probably a matter of coincidence only so far as the barometer alone is concerned. There is a larger number of patients streaking in March, May and especially June than in other months. Hemorrhage occurred more frequently in June than in any other month."

I have observed in my hospital work that hemorrhages at times occur in epidemic form, a large number of patients bleed at the same time in a ward. This may be explained by some intercurrent infection, especially influenza, causing pulmonary congestion. But psychic influences may also be at work.

Any of the above-mentioned factors may be the apparent exciting cause, but this is not true of the majority of cases. In my experience, a large proportion of hemorrhages, especially copious ones, begin when the patients have the least reason to expect them. It is the universal experience in sanatoriums that patients who have been kept under a rigorous rest cure may bleed. As was already mentioned, more than one-half the serious hemorrhages begin during the night, when the patient is resting in bed, or sleeping, and suddenly wakes up with cough, followed by a mouthful of blood. In patients with eroded bloodvessels or miliary aneurysms in the lungs, bleeding is apt to occur without any known provocative cause, and usually it cannot be prevented by any known means.

[1] Boston Med. and Surg. Jour., 1914, **170**, 564.

Diagnostic Significance of Hemoptysis.—It has been repeatedly stated that all patients with hemoptysis should be considered tuberculous and treated accordingly, until the bleeding is proved to be due to some other cause. But just because the vast majority of hemoptyses are due to tuberculosis of the lungs, when the blood is derived from some other source, it at times proves a serious source of error. There is left a wide margin of error when we attempt to follow this principle of considering every case of hemoptysis as tuberculous. Cabot among 3444 cases of hemoptysis treated at the Massachusetts General Hospital, found only in 1723, or 50 per cent, was the bleeding due to phthisis; Jex-Blake, in 54.6 per cent of 909 patients; and Stricker 77.6 per cent of 900 patients with a history of hemoptysis. Ware, among his private patients, observed 386 cases of hemoptysis among whom no less than 62 showed no evidence of disease, which would explain the occurrence of blood-spitting. Among the patients who consult me at my office, fully 50 per cent of those who have hemoptysis are not at all phthisical.

The most perplexing cases that present themselves in physicians' offices are patients who claim that several days ago they expectorated blood. In many the blood was derived from the nose, throat, gums, etc. Examination of these parts may not reveal any irritation, hyperemia or varices, while in the chest there are found some indefinite signs of an apical lesion which may be of non-tuberculous origin, or some changes in resonance and breath sounds indicating a perfectly healed lesion, thus leading to an erroneous diagnosis of active tuberculosis. This is especially seen in cases of epistaxis in which the blood trickled down the posterior nares, exciting cough productive of blood, or blood-streaked sputum. Some patients have epistaxis during the night, wake up spitting blood and present themselves promptly in the morning for a medical examination which does not reveal any definite clues as to the source of the bleeding.

Streaky Sputum.—Great care must be exercised before diagnosing tuberculosis based on a history of blood-streaked sputum. While this, when originating in the lungs, may, in rare cases, be a precursor of a large and profuse hemorrhage, it is, however, a fact that streaky sputum only rarely originates in the pulmonary parenchyma; in the vast majority it comes from the nose, throat and especially the bronchi. West[1] says that streaky hemoptysis is far more frequent in bronchitis than in phthisis. When it occurs in phthisis it is generally due to the same cause, viz., the rupture of distended capillaries in the bronchial tubes as the result of violent coughing; but when the tubes are the seat of tuberculous ulceration, bleeding may sometimes take place from the ulcerated surface, usually in small amount and streaky, but occasionally in larger amount. Individuals suffering from chronic rhinopharyngeal and subacute tonsillar inflammation of any sort at times

[1] Diseases of the Organs of Respiration, London, 1909, **2**, 381.

expectorate blood-streaked sputum. This occurs largely in the morning; while "clearing the throat" some mucus is expectorated showing streaks of blood. The patient is frightened, and with a view of convincing himself, begins to cough more strongly, finding on inspecting the material that it really does contain blood. The force used to dislodge the attached secretions may be responsible for the streaks of blood brought out. A careful examination of the throat may not show anything suggestive of the source of the blood.

In addition to rhinopharyngeal inflammatory processes there are other conditions of the throat which may produce hemoptysis. Among them may be mentioned certain new growths of the larynx, such as vascular fibromas, hemorrhagic laryngitis, etc. In several cases under my care these non-tuberculous conditions proved to be a source of error.

In many cases with a history of streaky sputum a diagnosis can only be arrived at by careful observation of the patient for weeks, after the presence or absence of fever, tachycardia, anorexia and physical signs in the chest are diligently studied. Very often the blood is derived from congestion in chronic pharyngitis with a spongy mucous membrane, or from dilated or varicose bloodvessels in the trachea, or main bronchi, common in asthma and chronic bronchitis. Varicosities of the esophagus are also said to be quite common. These "esophageal piles" may cause very copious hemorrhages. Many writers have described hemoptysis due to varices at the base of the tongue which are visible in the laryngeal mirror. The veins may be large and dilated and often extend to the fold of the epiglottis, or only a number of blue or dark blue specks may be noted, at times confluent, greatly resembling a vascular tumor. These are very often causes of hemoptysis. They are found mostly in persons between forty and fifty years of age, especially those who show stigmata of arteriosclerosis and other varicosities, as on the legs, or hemorrhoids.

These false hemoptyses have been described by many English physicians. Williams[1] speaks of persons who, without any symptoms of lung disease, bring up quantities of blood and recover without permanent cough. He says that they were generally middle-aged and often had the arcus senilis. Recovery is the rule. Several cases of this class come within the writer's observation annually. Sir Andrew Clark[2] also describes "arthritic hemoptysis" occurring in elderly persons free from ordinary disease of the heart and lungs; a form of hemoptysis arising out of minute structural alterations in the terminal bloodvessels of the lung. These vascular changes occur in persons of the arthritic diathesis, resemble the vascular alterations found in osteoarthritic articulations, and are themselves of an arthritic nature. More recently F. de Havilland Hall[3] attributed these hemorrhages to high vascular tension. Even though it occurs in a patient who has had

[1] Pulmonary Consumption, London, 1887, p. 135.
[2] Tr. Med. Soc. of London, 1889, **12**, 9; Lancet, 1889, **2**, 840.
[3] Lancet, 1915, **2**, 329.

phthisis, this form of hemoptysis is not necessarily due to a recrudescence of the disease, but may be the result of high tension with degenerate vessels.

At times persons suffering from pulmonary emphysema expectorate blood-streaked sputum, especially after paroxysmal cough. In rare instances I have observed emphysematous subjects expectorating pure blood—as much as an ounce or two. While in such cases we always suspect that we are dealing with the emphysematous form of fibroid phthisis (see p. 422), yet I have seen many cases in which subsequent observation, for a long period of time, has shown conclusively that no active tuberculosis developed.

Hemoptysis during Acute Respiratory Diseases.—We have already mentioned that acute rhinitis, pharyngitis, tonsillitis, etc., may be accompanied by the expectoration of blood. In fact, when a patient complains of hemoptysis and shows signs and symptoms of an acute affection of the upper respiratory tract the chances are greatly in favor of the blood being derived from the rhinopharynx and not from the lungs. Moreover, tuberculosis never begins with acute coryza, pharyngitis, or tonsillitis.

In *lobar pneumonia* the rusty sputum is characteristic. But in many cases the expectoration of pure, bright red blood is observed. In bronchopneumonia, hemoptysis is even more frequent, and during the recent epidemic of *influenza* a large proportion of patients in whom pneumonia complicated the process had more or less profuse hemorrhages. The differentiation is made by the history of the case, its epidemic occurrence, the symptomatology which is characteristic of influenza, and the location of the pulmonary lesions.

Hemoptysis in Pleurisy.—In many cases of pleurisy with effusion the onset is with a more or less copious pulmonary hemorrhage. I have met many cases in which after the bleeding ceased physical examination revealed an effusion into the pleura. In some phthisis developed subsequently, but others remained well for an indefinite time after the effusion was absorbed. I have also noted that this is more likely to occur in cases of *interlobar pleurisy*, first described by Dieulafoy. There may be blood-streaked sputum, and at times abundant hemoptysis, which may recur at variable intervals. After the interlobar effusion has been absorbed, or an abscess remains after an interlobar empyema, recurrent attacks of hemoptysis may occur. The differentiation of these cases from tuberculosis is discussed elsewhere in this book.

Hemoptysis in Heart Disease.—Blood-spitting in heart disease is often treated as of tuberculous origin with disastrous results. Inasmuch as we very often meet with cardiacs who are emaciated, cough, and have occasionally mild pyrexia, the diagnosis of tuberculosis is at times made erroneously. It is in fact usually supported by some physical signs in the chest, because cardiacs may show defective resonance, alteration in breath sounds, and even rales over an apex,

or other parts of the chest as a result of infarction, peripheral thrombosis, or brown induration. I have seen cases of organic heart disease treated in tuberculosis clinics and day camps in New York City for months. In infarction the expectorated blood may be bright red, and very copious in rare cases, but in mitral disease small, solid, purple or black lumps, which sink in water, are usually brought up. They are derived from ruptured capillaries in the walls of air cells, where they remain for some time before they are expectorated. The experienced eye can generally distinguish them.

According to Frederick W. Price,[1] mitral stenosis is probably the next most frequent cause of hemoptysis to pulmonary tuberculosis, and a common source of error. Among 3444 cases of hemoptysis in the Massachusetts General Hospital, R. Cabot[2] found that in 1177, or over 34 per cent, the bleeding was due to mitral disease. Perhaps the heart is not examined at all, or if it be examined it is by no means rare for the characteristic murmur to be absent. Furthermore, because there are frequently apical signs, as has already been indicated, phthisis is often diagnosed. In several cases I was nearly trapped by this similarity of mitral disease to phthisis, but noting some irregularity in the heart-beat, I investigated further and diagnosed mitral stenosis. It must always be remembered that while active phthisis is not altogether excluded with heart disease, yet it is extremely rare, especially in mitral stenosis.

In *aneurysm of the aorta* the end often comes through a rupture of the sac and fatal hemorrhage occurs. But in many cases streaky sputum is seen for weeks, or even for months, before the fatal hemorrhage finally kills. I have seen several cases in which pressure exerted by the aneurysm on the lung, or on a bronchus, produced signs simulating an apical lesion, and it is not exceedingly rare to find that cases admitted to and kept for months in sanatoriums are found suffering altogether from aneurysm of the aorta.

In *pulmonary infarction* hemorrhage is the rule. Mistakes of confounding these cases with tuberculosis may be avoided by a careful consideration of the history of the patient, an examination of the peripheral veins, the heart, etc. Still, many of these patients are often treated for tuberculosis because of the hemorrhage (see p. 552).

Hemoptysis in Bronchiectasis, Syphilis, and Cancer of the Lungs.—In bronchiectasis bleeding is not uncommon, and I have seen copious hemorrhages due to this cause. The blood is derived either from dilated and congested bloodvessels in the proliferated mucous membrane, or from inflammatory changes in the mucosa, or from small eroded aneurysms in the walls of bronchiectatic cavities, similar to those found in tuberculous excavations. As a rule, it is encountered in older persons, but recently, since bronchiectasis has become quite common as a sequel of influenzal bronchopneumonia, we find hemoptysis due

[1] British Med. Jour., 1912, **1**, 287.
[2] Differential Diagnosis, 1914, **2**, 433.

to this cause among younger persons very frequently. During the hemorrhage the diagnosis may be difficult, though a careful history clears up the case. In syphilis of the lungs, hemoptysis of various degrees has been encountered.

Hemoptysis often occurs in cases of *cancer of the lung*, and is at times a source of error in diagnosis. In the early stages of cancer of the lung the symptoms may simulate those of tuberculosis very closely. The bleeding, if it does occur, is usually very obstinate; the patient keeps on expectorating dark clots of blood. Pure, bright blood is rare at this stage. The differential diagnosis is discussed in its proper place (see p. 547). In advanced cancer of the lung there may occur copious pulmonary hemorrhages.

Other pulmonary diseases which may cause hemoptysis are fibrinous bronchitis, some cases of gangrene of the lung, echinococcus, pulmonary spirochetosis, and actinomycosis of the lungs. The differential diagnosis is discussed in Chapter XXVIII.

Hemorrhages from the Esophagus.—Varicosities of the esophagus, "esophageal piles," have already been mentioned as liable to cause hemorrhages which closely simulate pulmonary hemoptysis. In one case under my observation the bleeding was copious, almost threatening, and a diagnosis could not be made for some time. There have been reported cases in which the mucous membrane of the gullet was covered by enlarged, dilated, and tortuous veins. It is mostly found in persons suffering from cirrhosis of the liver. But it may occur in those who have no hepatic trouble. Patients suffering from cancer of the esophagus also may bring up blood with their expectoration; in the advanced stages of the disease the bleeding may be copious. The neoplasm may extend to, and perforate a bronchus, and the blood may thus be brought out through the trachea and larynx. The diagnosis should offer no difficulties to those who carefully examine their patients.

Menstrual Hemoptysis.—Phthisical women, if they are to have hemoptysis at all, are more apt to have it during the menstrual period. It has been observed that during menstruation there is usually an increased blood-pressure and congestion of the laryngeal mucous membrane, and some state that active periodical hyperemia of the lungs occurs at that time and this would favor extravasation of blood, especially in the affected area. According to Macht[1] these periodical hemorrhages, which may be very slight or profuse, may persist after the patient has improved in health and the tuberculous process becomes arrested. Periodic hemorrhages in consumptives at the time of menstruation may take place from other organs than the lungs. Thus, Wilson and Newman have reported such hemorrhages from the trachea and upper respiratory passages. Macht also reports a rather interesting case of a woman with pulmonary tuberculosis with intestinal complications—ulcer in the bowels—who regularly had severe hemorrhages from her intestines at her periods.

[1] Am. Jour. Med. Sci., 1910, **140**, 835.

Vicarious menstruation, which is very rare, appears to be due in most cases to tuberculosis. But in evaluating vicarious menstruation it must be borne in mind that amenorrhea is very frequent in phthisis, and in this disease hemoptysis is frequent; it is therefore not surprising that hemoptysis should occasionally occur while the menstrual flow has been delayed or suppressed.

Hemoptysis is apt to occur in pregnant tuberculous women periodically almost to an extent as to suggest that it is vicarious in character. On the other hand, non-tuberculous pregnant women have hemoptysis at times, especially if they cough severely for any reason. Many cases of this sort have come under my observation. After childbirth they usually cease bleeding if they are not tuberculous. The diagnosis in these cases is very difficult at times because incipient phthisis often improves during pregnancy and is thus liable to lead to a false sense of security. A careful examination of the chest and several microscopic examinations of the sputum will, however, clear up the case in most instances.

Several authors have also reported hemoptysis in women during lactation; soon after the infant is weaned, they stop expectorating blood. The causes of these hemoptyses are obscure.

Hemoptysis of Nervous Origin.—In hysterical individuals, especially women, we at times observe symptoms of incipient phthisis, including hemoptysis, but repeated physical examinations do not disclose any pathological changes in the lungs. Physicians of former generations have therefore spoken of "hysterical hemoptysis." In most of these cases we find that the blood is derived from the gums, or from the throat, brought out by violent cough. In their efforts to excite sympathy they are even apt to produce bleeding mechanically by injuring the buccal mucous membrane. When with this there is also cough, dyspnea, pain in the chest, and even fever, symptoms commonly found in hysterical subjects looking for sympathy, the diagnosis is at times very difficult. However, in addition to the absence of signs of a lung lesion, there are found positive stigmata of hysteria. On the other hand, the fact must not be lost sight of that hysterical individuals may become tuberculous, and that tuberculous individuals are often manifesting symptoms of hysteria. Indeed, some patients who have had one or more attacks of hemoptysis become obsessed with the fear for blood and consider themselves the most unfortunate among tuberculous patients. When told by the physician that their disease is progressing rather favorably, they often retort, "Why, doctor, I am a hemorrhage case." This is mostly seen in patients who have spent some time in sanatoriums and have either bled themselves, or observed copious, perhaps fatal hemorrhages in other patients. They constantly watch their expectoration for blood and may, during a fit of cough during the night, rise, light up the room and carefully inspect the sputum brought out with a view of finding a speck of blood. This fear for bleeding, which one author has called *hemophobia*, may dominate the entire

clinical picture, and it is at times difficult to manage this class of patients.

In certain diseases of the central, as well as the peripheral nervous system, hemoptysis may occur. Thus, in some cases of locomotor ataxia, cerebral hemorrhage, etc., hemoptysis is at times observed, though a careful examination of the chest fails to reveal signs of a pulmonary lesion. In some cases of epilepsy also it has been observed that the patients expectorate blood after a paroxysm. In these cases the blood may be derived from the tongue, which was injured by the teeth. It has, however, been shown that disturbances in the central nervous system may result in hemoptysis. Experiments by Brown-Séquard demonstrated that after injuries to the pons Varolii there were extravasations of blood into the lung tissue. François-Frank found that strong irritations of the peripheral nerves may result in bleeding from the lungs. Lichtheim, Claude Bernard, Longet, and other physiologists have confirmed these experimental findings.

It must, however, not be rashly concluded that tabetics who expectorate blood are not tuberculous. In most cases that came under my observation the tuberculous lesions were localized, or positive sputum was obtained. But it appears to the writer that the lung lesions are usually quiescent; active and progressive phthisis is rare in tabetics, perhaps because of the syphilitic substratum. In rare cases hemoptysis in tabetics was found to be distinctly non-tuberculous in character.

Hemoptysis of Unknown Origin.—We have already mentioned that every physician of experience has met with cases of hemoptysis showing no symptoms or signs of any disease to account for the bleeding. Very frequently we are consulted by patients in whom the most painstaking examination and clinical observation extending over a long period of time reveal no cause for the pulmonary hemorrhage. They remain healthy indefinitely. In some the hemorrhages are recurring at irregular intervals, and at times the amount of blood brought out may be considerable. The patient after losing considerable blood remains anemic for some time, but soon recuperates, and feels well indefinitely. Various suggestions may be made as to the origin of the bleeding, but none can be proved to the satisfaction of those who are competent to pass an opinion. Those who consider these pulmonary hemorrhages as of the same diagnostic significance as epistaxis are as safe in their assertions as those who are more explicit and careful in their diagnostic utterances.

I have met with several of this type of cases treated as tuberculous, kept in sanatoriums, or banished to distant climes. But they never developed symptoms of active pulmonary phthisis. Emanuel Libman and Reuben Ottenberg speak of *hereditary hemoptysis*. They have observed a case in which for four generations more or less copious hemorrhages from the lungs occurred at irregular intervals, and in no instance has phthisis developed. Similarly epistaxis is occasionally seen to run in families. With hemoptysis, however, there is always

danger that the patient will be pronounced affected with hereditary tuberculosis and treated as such, though in fact it is of no more significance than a nose bleed.

Some of these hemoptyses of unknown origin may be due to abortive tuberculosis (see p. 414). In others they are due to bronchiectasis which is not easily diagnosticated. In one case under my observation for eight years tuberculosis was diagnosticated and institutional treatment instituted; then other conditions were accused, but finally we made up our minds that it is due to multiple bronchiectatic cavities. The bleeding in this case occurs at irregular intervals, is nearly always copious and even threatening, the patient remaining exsanguinated, but soon recuperates. It seems that phlebotomy prevents the hemorrhage in this patient, or at least mitigates its severity.

Localization of the Source of the Hemorrhage.—Heretofore the determination of the side of the chest in which the bleeding takes place was merely of academic interest, because it made very little difference on which side the ice-bag, which has been traditionally used in the treatment of this symptom, was applied. But recently, since we found that an artificial pneumothorax may stop a copious hemorrhage after everything else has failed, it is of practical importance to localize the bleeding-point.

In cases which have been under observation for some time, and it is known that the lesion is unilateral, the problem may be simple, inasmuch as profuse bleeding usually implies a cavitary lesion. But in bilateral cases it is difficult, often impossible, to determine positively which lung is bleeding. Percussion must not be done for fear of increasing the bleeding, and auscultation may be of service in showing a limited area of moist, consonating rales, and perhaps amphoric breath sounds. But it is a noteworthy fact, which must never be lost sight of, that during profuse hemorrhages the blood may be aspirated into the non-bleeding lung and produce all sorts of rales. It is therefore, at times, impossible to decide positively which lung is bleeding.

In rare instances we hear murmurs, synchronous with the heart-beat, over the site of excavations. Gerhardt found that these murmurs originate in arteries which traverse the walls of cavities and he verified his observations at the autopsy table. In several cases this phenomenon was observed by me, the murmur was audible below the clavicle, and over the same area were most of the classical signs of pulmonary excavation. These patients are apt to bleed copiously, and they often succumb to a sharp hemorrhage. Here we know that the source of the bleeding is the branch of the pulmonary artery which traverses the cavity, and operative treatment (an artificial pneumothorax) may be attempted when a hemorrhage cannot be controlled otherwise. But these cases are rare and in the average case we cannot say with any degree of certainty that the bleeding vessel is located in a superficially recognized excavation, and not in another one, either located deeper, or altogether in the other half of the chest. I have repeatedly

seen cases in which after a copious hemorrhage the more affected side remained unaltered, while in the unaffected lung signs of a new lesion appeared.

According to Stricker,[1] the bleeding comes from an eroded vessel when it occurs suddenly during the course of acute and progressive phthisis, while in chronic cavitary phthisis it is usually derived from an aneurysmal dilatation of a vessel. Repeated hemorrhages accompanied by fever point to progressive decay of the affected area in the lung. Hemoptysis in the advanced stages of phthisis is derived from eroded arteries, and for this reason the prognosis is less favorable than in hemoptysis in incipient cases or in initial hemorrhages, which are, as a rule, of venous origin.

Differential Diagnosis.—In cases of initial hemoptysis it is imperative to ascertain whether the blood is derived from a tuberculous lesion or is due to some other cause. *It must never be lost sight of that hemoptysis may be a symptom of every disease of the upper and lower respiratory tracts, tuberculous as well as others.* Careful examination of the nose and throat may reveal that it is altogether due to congestion or varicosity of the mucous membranes of the upper respiratory tract, as has already been mentioned. When the sanguineous fluid expectorated is uniformly bright red and watery, it is, in all probability, derived from the mouth. In case no symptoms or signs of a pulmonary lesion are discovered, and the bleeding cannot be ascribed to a non-tuberculous condition, the heart is normal, and there is no history of an injury, the patient is to be placed under prolonged observation before deciding that he is not tuberculous. But it must always be borne in mind that *mere streaks in the sputum may be due to many causes other than tuberculosis of the lungs, and a diagnosis of phthisis should not be made because of their presence alone.*

In copious hemorrhage, when it is not feasible to examine the patient's chest carefully, it is often difficult to decide whether the bleeding is due to a tuberculous lesion, a bronchiectatic cavity, pulmonary syphilis or, in rare cases, whether it is not altogether *hematemesis*. The last-mentioned condition may simulate hemoptysis because the patient may have aspirated the blood into the respiratory passages and then expectorated it; while in hemoptysis the blood may be swallowed and then vomited. It may then greatly simulate blood derived from the stomach, viz., black or chocolate-colored, thick lumps or clots, mixed with the contents of the stomach, and the stools may subsequently show evidences of blood. I have met with cases in which the diagnosis could not be made immediately, and I have seen several tuberculous patients in whom ulcer of the stomach was diagnosed and they were operated upon. We may, however, be guided by the following points: In hemoptysis the blood is, as a rule, coughed up, bright red, frothy and mixed with sputum. It is also alkaline and

[1] Nothnagel's Handbuch d. spez. Pathol., **14, 7.**

does not clot. But many patients swallow the blood and then vomit it out; it is then acid in reaction. Auscultation may reveal rales in some part of the chest, and a careful history will show that the patient has been coughing, expectorating, etc., for a long time, while in cases of hematemesis the history points to disturbances in the gastric functions, and physical signs may be discovered in the abdomen. *In hemoptysis we invariably observe that after the cessation of active bleeding the patient keeps on coughing and expectorating clotted blood for several days, which is never observed in hematemesis.* But when the hemorrhage from either source is brisk and copious, and there is no history, the points just enumerated are often of little or no value, because the blood is bright red, alkaline, and not mixed with either sputum or gastric contents. However, such profuse hemorrhages are, as a rule, seen in advanced consumptives in whom there are always to be noted the stigmata of tuberculosis.

In cases in which the diagnosis has not been previously established, bleeding from the deeper respiratory passages may, in rare instances, be difficult of differentiation as to whether it is derived from a tuberculous lesion or from a *bronchiectatic cavity*. I have been guided by the pulse and temperature of the patient—when these are normal, and the general condition of the patient is good, the chances are that there is a bronchiectatic cavity, especially in persons over forty-five years of age. When physical examination shows that the lesion is localized in a lower lobe, while the apices are free from pathological changes, the disease is almost invariably non-tuberculous bronchiectasis, pulmonary abscess, gangrene, etc. In older persons with arteriosclerosis the so-called "arthritic diathesis" is to be thought of. Usually a careful history clears up the diagnosis, while in rare borderline cases we should reserve our opinion until the hemorrhage ceases and a careful examination of the patient is feasible.

In addition to tuberculosis the following conditions are liable to cause pulmonary hemorrhage: Cardiac disease, aneurysm of the aorta, hemophilia, bronchiectasis, syphilis, abscess, and gangrene of the lung, certain acute specific fevers, pneumonia, epidemic influenza, suppurative processes in the mediastinum, foreign bodies in the bronchi, injuries to the chest, paroxysms of pertussis, echinococcus, cancer, actinomycosis, aspergillosis, hydatid, bronchopulmonary spirochetosis, *distoma pulmonale westermanni*, and pneumokoniosis.

Prognostic Significance of Hemoptysis.—Patients, almost without exception, overestimate the significance of hemoptysis and are more terrified at the appearance of a speck of blood in their sputum than by any other symptom or complication of phthisis, excepting perhaps pneumothorax. It is for this reason that initial hemoptysis has been described by some authors as a salutary phenomenon, because it draws the attention of the patient to the condition of his lungs which he may have otherwise neglected. In fact, I have known cases in which hemoptysis was actually life-saving for just this reason in patients who had

coughed for months, and presented other symptoms of phthisis, all of which they considered a trifling affair, when, like the climax of a slowly developing drama, hemoptysis made its appearance, opening their eyes, or even those of their physicians, so that proper treatment was instituted.

A hemorrhage may prove fatal immediately or within a few days of its appearance; or, if the patient survives it, it may have an influence on the course of the disease.

Prognosis in Initial Tuberculous Hemoptysis.—We have already mentioned that in many cases pulmonary hemorrhage, even when due to tuberculous lesions, is not necessarily followed by symptoms of phthisis. Every physician has among his clientele patients who coughed out more or less blood years ago but have never suffered from any obvious disease of the lungs. "Outspoken tuberculosis does not necessarily follow hemoptysis," says Frederick T. Lord,[1] "which may occur in patients with apparent good health and sound lungs. In 1768, Goethe, at the age of nineteen years, and then a student at Leipzig, had an attack as follows: 'One night I waked with a severe hemoptysis and had enough strength and presence of mind to wake my room-mate . . . for several days I wavered between life and death.' For some months he thought he had pulmonary tuberculosis and must die young. At the age of eighty-two years he had hemoptysis again and died at the age of eighty-three years. His long and active life may serve as a comforting example to those who need encouragement. At the age of twenty-three or twenty-four years, Rousseau expectorated blood and gave up his work as a teacher of singing. He died at the age of sixty-six." A fatal issue in initial hemoptysis is extremely rare. I have never seen one.

Proportions of Deaths Due to Pulmonary Hemorrhages.—When profuse, the patient may be exsanguinated and succumb to cerebral anemia, or the blood may overflow the bronchial tree and suffocate him, especially when it occurs suddenly while the patient is asleep. While this outcome is seen now and then, it is a very rare occurrence. Louis had but 3 fatal cases in 300 consumptives; Williams 4 out of 198 fatal cases; Wilson Fox 4 out of 101; Moeller saw only 1 fatal hemoptysis during fifteen years' experience with consumptives; Wolff reports a lethal outcome 3 times among 1200 tuberculous patients (0.25 per cent); Winsch 1 among 200 (0.5 per cent); Thue, 13 times among 975 patients (1.6 per cent); Sorgo 14 deaths among 5800 consumptives (2.4 per cent) and among 2.16 per cent of his patients subject to hemoptysis. McCarthy reports that at the Boston Consumption Hospital 400 deaths occurred during a period of two years, only 7 of which were due to hemorrhage. Lord reports that death as an immediate result of bleeding occurred in only 1 of 76 patients with hemoptysis at the Channing Home, and 2 of 142 at the Massachusetts General Hosital.

[1] Diseases of the Bronchi, Lungs and Pleura, Philadelphia, 1915, p. 360.

Death as a consequence of extension of pulmonary infection for which the hemorrhage was responsible, occurred in 1 case at the Channing Home, and 6 other cases at the Massachusetts General Hospital.

Williams reports that in 1000 cases, including 63 fatal ones where the patients had hemoptyses of one ounce and upward on one or more occasions, the average duration was seven years and six months; an average differing only by a few months from that of the total deaths. In 200 living cases of similarly extensive hemoptysis, the average was eight years and three months—about the same as that of the living cases generally. "It is only in the far-advanced stages that it is likely to curtail the duration of the disease. In early cases hemoptysis is comparatively unimportant," concludes Williams. *When we say that hardly one out of a thousand deaths due to tuberculosis is caused directly by hemorrhage, we are as near the true figure as possible; hardly 2 per cent of tuberculous patients who bleed succumb to this accident.*

Influence of Hemoptysis on the Course of Phthisis.—The influence of hemorrhage on the course of the disease is misunderstood by the average patient and often overestimated by the physician. It may be said that so long as it does not prove fatal immediately, and this is rare, as we have just shown, it has no effect on the patient nor on the disease. Many older writers have stated that it often has a rather salutary effect, and not altogether without reason, as is proved by the course of many cases subsequent to hemorrhages. Lebert, Flint, Wilson Fox, and others state that hemorrhages may produce a sense of relief, and cough and expectoration previously existing may temporarily disappear. Williams says: "To many patients its occurrence seems beneficial rather than otherwise, for the congestion is thus relieved and the system not materially weakened by the loss of blood." *I have seen many cases in whom the disease took a turn for the better, soon after a more or less profuse hemorrhage, and others in which the cough, anorexia, pains in the chest, etc., were ameliorated or disappeared after this accident.* We know that slight abstraction of blood is often beneficial inasmuch as it stimulates the blood-forming organs to produce more blood cells.

The fear, formerly entertained, that the blood, spreading all over the bronchial tree, is apt to inoculate new areas and produce new lesions in hitherto unaffected parts of the lung is now known to be without sound foundation, because reinfection is difficult or even impossible in the vast majority of cases. To be sure, we find that the bronchi contain blood while auscultating a patient during, or immediately after, a hemorrhage, but this is usually transitory, disappearing by absorption or expectoration within a few days after the bleeding ceases, and the original pulmonary lesion, if not progressive, remains the same as it was before, pursuing the same course as if no such accident had occurred. Cases in which after a hemorrhage a quiescent lesion begins to pursue an acute or subacute course and tuberculous bronchopneumonia is found at the autopsy are, in all probability,

due to a sudden reduction in the powers of resistance, about the causes of which we know nothing at present. They do occur now and then, but when taken in connection with the large number of hemoptyses in which this sequel does not occur, they are comparatively rare. In fact, H. J. Corper[1] found experimentally that bleeding an animal for days before, and continued throughout the period of tuberculous infection has no appreciable effect upon the tuberculous process.

The fear of bronchopneumonia as a sequel to pulmonary hemorrhage, entertained by many physicians, is not founded on fact. In afebrile patients, soon after the hemorrhage ceases, the temperature may be elevated for a few days, but within a week or so, after the effused blood is absorbed, the temperature comes down to the level at which it was before this accident. Hemorrhages occurring in febrile patients, at times, have the effect that after the cessation of the bleeding the patient is afebrile, as I have seen in many cases. On the other hand, many patients running high fever, when attacked by copious hemorrhage, continue with pyrexia after the hemorrhage ceases, and finally succumb to the active tuberculous process, or, more commonly, to miliary tuberculosis. How rarely bronchopneumonia follows pulmonary hemorrhage can be seen from figures published by C. G. Reinhardt Goodwin:[2] Among 1000 odd cases admitted to the sanatorium under his care in ten years, only 1 case of this kind has been recorded.

More than sixteen hundred years ago Galen stated that the prognosis of pulmonary hemorrhage depends on the fever which is apt to accompany it—afebrile cases recover, while in febrile cases the prognosis is gloomy. More extended experience in recent years has confirmed the opinion of this ancient and empirical clinician.

In hemoptysis the immediate, and especially the ultimate, prognosis depends less on the bleeding, its abundance or even repetition, than on the extent of the pulmonary lesion and the symptoms which accompany or dominate the clinical picture, the subsequent course of the original disease—phthisis—and the complications which may arise. When we find during a hemorrhage that a patient has a good, full pulse, less than 100 in frequency, and no fever or dyspnea, the immediate prognosis is good. If there are several repetitions of the hemorrhage during the subsequent few days, the prognosis is, as a rule, favorable so long as the pulse is good and there is no fever. Even fever is of no grave significance if it lasts but a couple of days. It is then due to absorption of the blood remaining in the bronchi. It is only when the fever is high and persistent for several days that it assumes serious import.

In case the pulse becomes small, soft, compressible, and rapid, we may be sure that the bleeding continues even if we do not see it brought up in large quantities through the mouth, *for we may have internal hemorrhage in phthisis*, the blood being retained in a large cavity,

[1] Tr. Am. Tubercul. Assn., 1920, **16**, 360.
[2] Practitioner, 1917, **99**, 288.

while the feeble patient is unable to force it out by cough. This is especially apt to occur after large doses of morphine have been administered, or in severely emaciated persons.

In cases which had been active before the onset of the bleeding, having had fever, tachycardia, emaciation, etc., the prognosis after cessation of the bleeding is usually the same as it would have been had there been no such complication. The temperature usually drops during a brisk hemorrhage, but it rises again and the course of the diseases continues unabated. But if the temperature has been normal, or only slightly elevated, and the pulse is less than 100, full and bounding, the patient has a good appetite, and sedative drugs are judiciously, if at all, administered, the immediate as well as the ultimate outlook is indeed good.

In most cases the findings on physical exploration of the chest after moderate hemoptysis remain the same as they were before that event, although on auscultation we usually hear moist, consonating rales which may not have been there before the onset of bleeding. These rales may persist for several weeks. In some cases we find that the area of dulness over the upper lobe extends because of caseous or necrotic changes engendered during the hemorrhage. This dulness may disappear after the clots have been absorbed, or after the resolution of the pneumonic areas. More frequently it is in time supplanted by tympany due to excavation.

CHAPTER XI.

SYMPTOMS CAUSED BY DISTURBANCES IN THE GASTRO-INTESTINAL TRACT—THE SKIN—THE JOINTS.

GASTRO-INTESTINAL SYMPTOMS.

Frequency.—Some authors have stated that phthisis develops mostly in individuals who have been naturally bad eaters; others have maintained that those suffering from gastric derangement are most likely to fall prey to the disease, and Grancher says that "all consumptives have been, are, or will become dyspeptics." In practice we meet with many patients who have been treated for gastritis for a long time until the true nature of their disturbance became evident. The diagnostic, and especially the prognostic, significance of anorexia or gastritis in a disease which depends in its origin and outlook on proper nutrition, cannot be overestimated.

As far back as 1826 Wilson Philip[1] drew attention to the fact that many cases of phthisis are preceded for some time by severe indigestion. In his excellent monograph on the "Dyspepsia of Phthisis," W. Soltau Fenwick[2] quotes Todd, Sir James Clark, Budd, Bennett, Ancell, and other writers of the first half of the nineteenth century, to the effect that dyspepsia is a very frequent forerunner of phthisis. In those days some authors spoke of "gastric phthisis," and "pre-tuberculous dyspepsia" is even now mentioned by many writers. There is no doubt that incipient phthisis, as we know it at present, was in those days not recognized, and evidently this has been responsible for the notion that phthisis is often preceded by dyspepsia.

Recent investigations, however, do not confirm that gastro-intestinal disturbances are *per se* predisposing factors in the evolution of phthisis, though Fenwick says that, for his own part, he is quite convinced that there does exist a variety of dyspepsia which is peculiarly apt to be followed by pulmonary tuberculosis.

As an early symptom of phthisis, dyspepsia is quite frequent. Hutchinson[3] found it in 92 per cent of his cases, and in 55 per cent it was quite severe; Levison,[4] in 74.6 per cent; Mohler and Funk,[5] in 64.6 per cent of 1000 consecutive cases. Samuel Fenwick, Dobell, Pollock, and others have found it in nearly similar proportions. W. Soltau

[1] Treatise on Indigestion, London, 1826, p. 323.
[2] Dyspepsia of Phthisis, London, 1894.
[3] Medical Times, 1855, **1**, 583.
[4] Ohio State Med. Jour., May, 1905, **1**, 204.
[5] Am. Jour. Med. Sci., 1916, **152**, 355.

Fenwick states that "dyspeptic phenomena of sufficient severity to attract the attention of the patient are encountered in about 70 per cent of all cases of early phthisis, but that the early development of the disorder in any individual case depends to a great extent upon the sex of the patient, the type of the tubercular disease, and the previous condition of the digestive organs." He found that it is more apt to occur in females than in males, and, in general, in that variety of phthisis which commences insidiously and progresses slowly.

More recent investigations have only partly confirmed the findings of the above-mentioned clinicians, and there are writers who consider anorexia, though not a result of gastritis, a constant symptom of incipient phthisis, like fever, cough, nightsweats, emaciation, etc. An analysis of 3007 cases in the Phipps Institute[1] showed that 55.3 per cent presented symptoms referable to the stomach. It appears, however, that these gastric disturbances were in no way due to changes in the stomach peculiar to tuberculosis itself, the changes being such as might occur in any chronic wasting disease. Janowski[2] reports that among 700 patients, 35 per cent suffered from gastric disturbances, which were more often encountered in women than in men. With this Kuthy is also in agreement. He found that in 37.3 per cent of his male patients there were gastric disturbances, as against 50.1 per cent in his female patients. In the first stage, 38 per cent; in the second stage, 46.4 per cent; and in the third stage, 57.2 per cent showed these symptoms.

Symptomatology.—*One of the characteristics of the anorexia of phthisis is that, unlike the appetite in other diseases, it is independent of the fever, in many cases.* Many patients with but slight fever have an almost complete antipathy for food, while others, who have moderate fever, preserve an excellent appetite. Laségue said, "All patients who eat and digest their food well, despite having fever, are consumptives." In acute pneumonic phthisis, which is often difficult to differentiate from lobar or lobular pneumonia, I have placed great reliance on this symptom: In pneumonia the anorexia is invariably complete, while in acute phthisis the appetite may be retained more or less, and in spite of a temperature of 103° or 104° F. the patient is apt to ask for nourishment.

In incipient phthisis the appetite is often very capricious. One day a certain food is preferred, while the next it is despised. Morbid cravings are not uncommon, especially in women. A large proportion of patients cannot tolerate certain kinds of food—some will not eat meat, others refuse milk, eggs, etc. It seems to me, however, that the repugnance for milk and eggs is often not the result of the tuberculous process, but is an acquired characteristic, due to the stuffing with these articles of food which is so commonly carried to an extreme degree. Following the usual advice, "plenty of milk and eggs," is likely to

[1] Tr. Nat. Assn. Study and Prevent. Tuberc., 1910, **6**, 193.
[2] Ztschr. f. Tuberk., 1907, **10**, 493.

ruin an excellent appetite, if carried to extremes. Two or three quarts of milk, and a half or one dozen raw eggs daily, which tuberculous patients often consume, may result in a strong repugnance to these articles.

An aversion to fats of any kind is very frequently observed in phthisical patients. Hutchinson noted this fact over sixty years ago, and stated that 71 per cent of his phthisical patients disliked fats; 33 per cent could take them in but small quantities; while only 5 per cent liked them. Fenwick noted a marked aversion to fat in 64 per cent, and many of his patients developed this peculiar antipathy many months, or even years, before the onset of the pulmonary disease. He observed that among families which exhibit a marked predisposition to tuberculosis, it is not uncommon to find that several members possess a strong aversion to all forms of fat, and are often unable to partake of even a small quantity of this material without suffering from acidity, nausea, or attacks of biliousness. Occasionally we meet with tuberculous patients who dislike carbohydrate, especially saccharine, foods, the ingestion of which causes more or less severe gastric discomfort.

In many cases the anorexia improves with the improvement in the local condition in the lung; but we also meet with cases in which the tuberculous lesion is slowly progressing or quiescent, but the appetite improves, as if the organism had adapted itself to the tuberculous toxemia. In fact, almost insatiable hunger may be seen in rare instances.

In the early stages of phthisis digestion is fair, or good, in most cases. Indeed, it appears to me that *digestion in phthisis usually depends on the condition of the gastro-intestinal tract before the onset of the lung disease.* As was already intimated, the excessive quantities of milk and raw eggs may be responsible for the symptoms of dyspepsia in many cases, such as pyrosis, belching, flatulence, bad taste in the mouth, etc. The fact that these symptoms may be relieved by appropriate corrections in the diet is in favor of our contention. Excepting in advanced cases, and in alcoholics, vomiting, if it occurs at this stage, is due to cough, as has already been detailed when speaking of the emetic cough. In the advanced cases it is likely to be preceded by nausea, which is not the fact with the emetic cough. In many early cases symptoms of *vagotonia* are quite prominent and they may dominate the clinical picture. There may be heartburn, pain after meals, pyrosis, belching, salivation, hunger pain, etc. Yet an examination of the stomach and its functions may not reveal anything to account for these symptoms.

Causes of Anorexia.—It appears that the anorexia of phthisis is of toxic origin. Analyses of the gastric contents have not revealed any constant changes in the anatomy or functional activity of the stomach in the early stages of phthisis. In some cases hyperchlorhydria is found, in others hypochlorhydria, while in many others the free and combined acids remain in about normal proportions. Nor have any

constant secretory or motor disturbances been observed. The physiology and pathology of the stomach in early phthisis, as studied by Klemperer, Hayem, Einhorn, Brieger, Fenwick, and others, show no characteristic functional changes.

Many French authors, notably Marfan,[1] are of the opinion that the gastric symptoms in early phthisis are due to the general anemia which causes sluggish secretion of gastric juice, weakness of the smooth musculature, and hyperesthesia of the gastric nerve endings of the vagus. Fenwick, finding that the dyspepsia in phthisis is not a direct result of pyrexia, nor of direct irritation of the mucous membrane, concludes that it is probably due to the chronic absorption of certain toxic substances which are manufactured in the pulmonary cavities; but he describes a form of dyspepsia which often precedes the development of pulmonary tuberculosis, when cavities are out of the question.

The gastric symptoms appear to be analogous with those observed in chlorosis, and the severe anemias, which cause ischemia of the digestive tract. But, as Janowski points out, many tuberculous patients without any anemia also suffer from gastric symptoms, and he concludes therefore that the anorexia is not invariably due to general anemia, but to ischemia of the gastric and intestinal mucosa. This explains why so many different results have been obtained from analyses of the gastric contents. It is the paroxysmal proclivity of the gastric disturbances which is characteristic of early phthisis. It is also a fact that reported investigations of gastric functions do not discriminate, as a rule, between early and advanced, febrile and afebrile, acute and chronic, active and quiescent lesions. Hence the great differences in their findings.

Gastric Symptoms in Advanced Phthisis.—The anorexia and other gastric symptoms of early phthisis usually subside in cases pursuing a favorable course and the patients recover. But in cases with progressive disease, especially those characterized by pulmonary excavations, more or less severe symptoms of dyspepsia are present. Nearly a century ago Louis found that about two-thirds of his phthisical patients had shown signs of dilatation of the stomach. W. Soltau Fenwick found among 100 autopsies in cases of tuberculosis in which he took special notes on this point, that the lower margin of the viscus extended below the level of the navel in 64, and he says that it is rare while performing an autopsy on a phthisical subject to fail to encounter some increase in the dimensions of this viscus. The degree of gastrectasis appears to bear a direct relation to the extent and chronicity of the pulmonary lesion. Of course, this gastrectasis may be pretuberculous—we have already shown that those with the asthenic constitution are more likely to contract phthisis than others. But it may also be one of the results of tuberculous toxemia which is effective in producing atony of muscles—both striated and smooth.

[1] Troubles et lesions gastriques dans la phtisie pulmonaire, Paris, 1887.

Chronic catarrh is very frequent, but true tuberculous ulcers are exceedingly rare, probably because the stomach contains comparatively little lymphoid tissue, and bacilli cannot reach there through this channel, and the acid secretions are inimical to the growth of tubercle bacilli. Fenwick, after a careful search, was able to discover the records of 24 cases of this affection, several of which are, however, open to suspicion; while among the notes of 2000 necropsies on cases of phthisis performed at the Brompton Hospital he could find only 2 instances in which tuberculous ulcers of the stomach were discovered. Lauritz found 4 cases of undoubted tuberculous ulcers in the stomach among 580; Melchior 6 in 848 autopsies, and Gassmann 6.13 per cent in 600 autopsies. Mohler and Funk did not find a single instance of gastric ulcer in 85 autopsies, and in 128 consecutive autopsies made at the Montefiore Hospital on cases under the writer's care only one case of tuberculous ulceration of the stomach was discovered. There have been reported cases of perforation of tuberculous gastric ulcers into the peritoneum, though this is exceedingly rare because of the inflammatory adhesions which usually form around the ulcers and the peritoneum. Simple gastric ulcers are not infrequently found at autopsies on tuberculous bodies, but the proportion is not higher than among patients who succumbed to any cause.

In the vast majority of cases of advanced phthisis the appetite is poor; those who do attempt to eat usually display various distastes for certain foods, and even this is not constant—the appetite is often very capricious, and many develop morbid cravings. This is one of the reasons for difficulties in feeding phthisical patients in sanatoriums and hospitals. At times we meet with patients who retain an excellent appetite to the end and instances of bulimia are not unknown. Pain after eating, pyrosis, belching, etc., are very common, and vomiting is, at times, a prominent symptom. But while the emetic cough may be encountered in advanced cases, the vomiting at this stage is usually not of this type. They simply vomit because of gastritis, or dilatation of the stomach. This type of vomiting is usually preceded by nausea, belching, etc., and not by cough as in those having the emetic cough. Nausea and retching may persist for several hours after the vomiting, and the ejecta consist of sour food mixed with mucus. I have met with cases in which no food could be retained owing to vomiting, and some even with hematemesis. The prognosis in these cases is gloomy indeed.

In hectic cases the gastritis is often very troublesome and, combined with vomiting, nightsweats, cough, diarrhea, etc., it is one of the terminal symptoms of phthisis. In many cases, however, the pulmonary symptoms overshadow the gastric phenomena, but very often the latter are sufficiently pronounced to require great care and attention. The amyloid liver often contributes considerably to the digestive disturbances, and lardaceous changes in the bloodvessels of the stomach are not unknown. I have met with cases of this type, extremely

emaciated, hardly able to move a limb, yet they asked for food which, when given by the nurse, was relished with an apparently voracious appetite.

It appears that the dyspepsia of advanced phthisis is usually associated with pulmonary excavation, and is mainly caused by the prolonged intoxication characteristic of progressive and advanced disease. A fruitful source of gastric derangement is swallowed sputum, more common in women. The sputum not only irritates the mucous membrane of the gastro-intestinal tract, but some of it is also absorbed and produces toxemia. The mucous membrane of the gastro-intestinal tract eliminates poisons from the blood, which in their turn irritate these membranes, as is the case in acute mercurial poisoning in which mercurial albuminates circulating in the blood are eliminated into the intestines where they cause severe diarrhea. The injection of large doses of tuberculin may also cause diarrhea.

Intestinal Symptoms.—During the incipient stage of phthisis the bowels are unaffected in most cases, though we meet with constipation in a considerable proportion. But I doubt whether the proportion is higher than among people with modern habits of life, and dietetic conditions. In some cases the constipation is due to the sedative medication used for the control of the cough.

Diarrhea may be one of the symptoms of incipient tuberculosis. It is met with mainly in patients at the two extremes of life—in children under ten years of age and in senile patients. In children the diarrhea may be the only symptom, while examination of the chest may show nothing conclusive, or signs of tracheobronchial adenopathy may be found. In aged patients who have felt quite well, even claiming that they have not coughed, a chronic and persistent diarrhea should be considered a sign that a careful examination of the chest is urgent. It will be found that there are signs of old phthisical lesions in the lungs, and the sputum may contain numerous tubercle bacilli. Very rarely diarrhea is one of the symptoms of incipient phthisis in young adults.

In some patients the functions of the bowels remain more or less normal through the course of the disease, but this is rare. In most cases diarrhea makes its appearance with the advance of the disease. While in many cases it is due to tuberculous ulceration of the bowels, there are others in which it is caused by intestinal catarrh, very frequently the result of dietetic errors. In many the ingestion of large quantities of milk is responsible and eliminating milk from the diet promptly gives relief. In others the excessive amount of fat, mainly eggs, is responsible. Elsewhere it is pointed out that raw eggs are very frequently the cause of diarrhea (see Chapter XLI). Persons who have had intestinal trouble before the onset of phthisis are more liable to suffer from catarrhal diarrhea than others. As will be pointed out later when speaking of tuberculous ulceration of the intestine, the

differential diagnosis is exceedingly difficult. The prognosis depends on the causation of the diarrhea. When due to amyloid degeneration or tuberculous ulceration of the intestines the prognosis is grave.

EMACIATION.

Emaciation is a cardinal symptom of phthisis; one of the triad mentioned by Richard Morton, the others being cough and fever. Popular lore, as well as medical experience, have always associated tuberculosis with emaciation. Phthisis (Greek, φθισις), consumption, has its equivalent in every European language. That it is mainly due to the tuberculous toxemia, engendered by the metabolism of the tubercle bacilli, is evident from the fact that experimental tuberculosis is always accompanied by emaciation of the animals.

In acute galloping consumption, and in miliary tuberculosis, the emaciation is progressive and frightful, much more rapid than in other febrile diseases, as pneumonia, typhoid, etc., and this is one of the most important points in the differentiation of acute tuberculosis from other acute diseases. In children, when during or after an attack of measles, pertussis, etc., the wasting becomes very marked and there is dyspnea, rapid pulse, etc., acute tuberculosis is to be suspected.

While the malnutrition and wasting in phthisis are often caused, and always enhanced to a certain extent, by the gastro-intestinal disturbances which are concomitants of the disease in all its stages, we meet with emaciation almost constantly in active disease with fair gastro-intestinal functions. Some authors are inclined to attribute the emaciation to the lowered powers of absorption caused by a congenital narrowing of the lymph channels in the intestinal tract which is said to predispose to phthisis. But this has not been proved.

Extent of Emaciation.—Not only does the subcutaneous adipose tissue waste, but the nitrogen-containing muscles also vanish with astonishing rapidity. It is noteworthy that *the first muscles to waste are those of the thorax*—the pectorales, the scapular, the intercostals, etc. In many incipient cases we see a striking contrast between the wasted and flabby muscles of the chest—and in women occasionally the wasted breasts—and the fairly preserved contour of the muscles on the extremities. Moreover, the muscles and subcutaneous tissue of the affected side of the chest waste earlier than those on the opposite and unaffected side. The result is that the supraclavicular and supraspinous fossæ are more or less deeply excavated. This characteristic of the muscular wasting has recently been made available for diagnosis by the excellent studies of Pottenger. In some early cases the face remains full and is thus apt to deceive as to the state of nutrition of the patient whose trunk and abdomen are markedly emaciated.

Effects of Emaciation.—The weakness, weariness, loss of strength and vigor of the consumptive are greatly due to the muscular atrophy even in the early stages of the disease, and one of the best signs of

improvement is the regression in the muscular atrophy. There appears to be a direct relation between emaciation and the course of the disease. With each extension of the process in the lung, with each hemorrhage, there is a loss in weight, and with each improvement there is a gain in this direction, while in quiescent cases the weight remains unaltered. It may be stated that, with some exceptions to be mentioned later, the scale readings may be taken as a fair index of the evolution of phthisis, and when we consider it in connection with the temperature curve, we can follow the case, and interpret it from the prognostic standpoint, with a fair degree of safety.

There are, however, exceptions: Patients in whom the disease has been arrested, *i. e.*, in whom a quiescent lesion is smouldering, are apt to remain underweight indefinitely, though they feel quite well, and are more or less efficient.

When patients are progressively losing it is not advisable to tell them the extent of their malnutrition. The discouragement often pulls them down much further. Conversely, it is often observed that patients gain weight after changing their physician, entering a new sanatorium, etc., and thus gain a false impression that they are on the road to recovery. But after the novelty of the new surroundings has worn off, the gain ceases. They may then even lose progressively, and finally weigh less than before admission to the institution. To be of favorable prognostic significance, gain in weight must be persistent for several months.

In some cases of phthisis the emaciation is rapid and extreme; within a few months the body of the victim is reduced to a skeleton. These are the cases in which the disease runs an acute and progressive course—galloping consumption. Now and then we meet with patients in whom the disease is chronic, lasting for many years, still the emaciation is severe; the ribs, robbed of their adipose covering, protrude between the atrophied intercostal muscles so that we are unable to adapt the bell of the stethoscope to the chest. This cachectic form of phthisis is mostly seen in old people, and, inasmuch as they have no fever and hardly cough, latent cancer is at times erroneously diagnosticated.

Prognostic Significance of Emaciation.—Sanatoriums advertising their advantages usually show the average number of pounds gained by the patients during a certain period, and patients usually gauge their progress by the scale. This is correct in the vast majority of cases. An improving patient is one who gains in weight, and one who loses progressively is doomed. But to this there are exceptions. Gains in institutions, while the patient is under a rigorous rest cure and overfed for long periods, are good as far as they go. But in order that the patient should be pronounced improved, or cured, it is necessary that he should hold his gain after he becomes active at his occupation or at some other vocation which suits him. In this regard, the graduated labor system of Paterson at Fromley is superior to other forms of

institutional treatment. The gains attained at Fromley are said to be more lasting than those in the institutions where the inmates lead a lazy or indolent life. Similarly, patients who are treated at home, and allowed to do some work while under treatment, are more likely to keep their gains than the former class.

We must be careful in evaluating gains in weight. Sometimes the patient keeps on gaining moderately while the disease is progressing and we wonder why this is so. A careful investigation may show that the lower limbs are edematous, and it is not fat and flesh which is responsible for the increase in weight, but dropsical fluid.

At times we meet with patients in whom the lesion in the lungs is improving or stationary, and they have a good, or even a voracious, appetite, yet they keep on losing in weight. This is usually due to intestinal tuberculosis in which there may not be the characteristic diarrhea. This is a diagnostic point worth remembering, because it is often very difficult to decide whether the intestine is implicated in the process, and the prognosis depends so much on the condition of the bowels.

Seasonal Influences.—The seasonal influences on the weight of consumptives are best studied in sanatoriums. It appears that there are significant differences in this regard. At North Reading, Mass., Burns[1] found that the minimum amount of weight loss occurs in the colder months; the maximum loss occurs in the warmer months; and rapid increase in amount of emaciation appears during the spring months. Going hand-in-hand with this is the fact that deaths in July outnumber other months. At the Adirondack Cottage Sanitarium, Brown[2] found that the weight curve in pulmonary tuberculosis, if not influenced by change in climate or some other factors, rises from August to Christmas (sometimes to November), remains more or less stationary with minor fluctuations from Christmas to Easter (March), and sinks gradually from Easter to August. Brown adds that this corresponds closely to the normal weight curve. In Pennsylvania, Karl Schaffle finds the gains most marked during the fall and winter months. Among private patients in New York City I find that the summer months are not conducive to gains in weight, nor are the autumn months with their variable weather; but during the winter, especially during very cold seasons, the gains are extraordinary; even patients who are running low for one reason or another often gain somewhat, or remain stationary, during December, January, and February.

This is not true of other climatic regions. In a careful study of the weights of consumptives in eight sanatoriums in Denmark, N. S., Strandgaard[3] found that weekly weighing shows low gains during the winter and spring months from December to May. Then there is a distinct rise during the summer months, June, July, and August, reach-

[1] Boston Med. and Surg. Jour., 1914, **170**, 564.
[2] Osler's Modern Medicine, **1**, 380.
[3] Beitr. z. klin. d. Tuberk., 1914, **32**, 179.

ing its maximum in September, and declining in October, and more so in November and reaching its minimum in December. This is the exact opposite of conditions in the United States.

The subject deserves careful study in connection with meteorological conditions.

Fat Consumption.—The term "fat consumption" may appear incongruous, but we meet with cases of active phthisis in which the panniculus adiposus is well preserved, or even with excessive obesity, the *phthisiques gras* of some French writers. I see several cases of this sort annually in my private and hospital work. They appear healthy, with florid cheeks and well-formed bodies, and their only trouble is that nobody believes they are tuberculous. They cough and expectorate, often profusely, quantities of sputum with numerous tubercle bacilli, run a mild subfebrile temperature, at times have nightsweats. Many have more or less profuse hemoptysis and in two that were under my care the cause of death was copious terminal hemorrhage.

When these patients present themselves for examination one is loath to make a diagnosis of phthisis even when physical exploration of the chest reveals a typical lesion in one or both lungs, or cavitation; which is not uncommon. The course of the disease is rather slow; we may follow them for years without noting any marked changes in their general condition despite the fact that the lesion in the lungs is progressing and excavations are forming. Of course, only positive sputum findings are convincing to some patients or even physicians.

The obesity is mostly seen in female consumptives during or soon after the menopause, though I have met it in males, especially alcoholics and those having a history of syphilis. They usually have a voracious appetite and when told that they must eat well, they follow directions, often overdoing it. Combined with the rest which is urged and implicitly obeyed, the overfeeding is effective in producing fat, despite the activity of the disease. In tuberculosis implanted on pulmonary emphysema, and also in fibroid phthisis, the weight of the patients is often above the average, though real obesity is observed only rarely.

Fat consumption is also observed in children, especially infants of tuberculous stock. They appear well nourished and fat, but when we examine their muscles we find them flaccid and soft. These "pasty" infants have no resistance against infection, and are carried off by any acute disease which flares up the dormant tuberculous lesions. Similarly, tuberculous meningitis and bronchopneumonia are often seen in rather fat children.

THE SKIN.

In addition to the wasting of the muscles and subcutaneous fat, atrophy of the skin is one of the early changes in phthisis, first described by Clarence L. Wheaton,[1] of Chicago, and then by Pottenger.

[1] Jour. Am. Med. Assn., 1910, **54**, 2123.

On inspection it is noted that the skin over the site of the lesion is thin and the subcutaneous tissue vanished. According to Pottenger, this is part and parcel of the general degeneration, and occurs after the process has existed for some time. It denotes chronicity rather than earliness, although it is often found over comparatively early tuberculous processes. In such cases it may be presumed that there was an old quiescent lesion which has become the seat of renewed activity.

The complexion of the consumptive is usually pale, though at times we meet with patients advanced in the disease who have retained a florid color. In some the hectic flush is evident at first sight; it is mostly seen at the time when the daily rise in temperature occurs. Occasionally this redness appears only on one cheek, corresponding usually to the affected side of the lung, as is discussed elsewhere. In

FIG. 45.—Tuberculides. (Ormsby.)

fibroid phthisis, and in those with emphysema, in the advanced stages of which dilatation of the right heart occurs at times, there may be cyanosis of variable degree. Cyanosis is also a cardinal symptom of miliary tuberculosis. In many cases with extensive excavations in both lungs there is hardly any cyanosis, at most some livid tint of the lips may be elicited on careful observation, but in fibroid phthisis cyanosis is frequently marked. In far-advanced disease with amyloid changes, the skin shows the characteristic appearance of this condition. According to Meyer Solis-Cohen[1] between 25 and 33 per cent of tuberculous patients exhibit flushing, burning, sweating, urticaria; between 14 and 25 per cent subjective sensations of heat, angioneurotic edema, dermographia, etc., all of which he attributes to autonomic disturbance.

[1] Am. Revue Tuberc., 1917, **1**, 289.

Chloasma Phthisicorum.—Smooth, shining, and non-desquamating, yellowish-brown spots are occasionally seen quite early in the disease on the forehead and upper parts of the face. They are frequently single, but often confluent, forming large patches which in female patients may be a great source of annoyance. My experience with consumptives confirms the observation made long ago by Jeannin to the effect that chloasma phthisicorum is mostly seen in connection with enlarged glands, and that these patients only rarely suffer from hemoptysis. In fact, I have looked in all cases of hemorrhage that have come under my observation during the past five years and found

FIG. 46.—Acnitis. Dr. Quinn's case. (Ormsby.)

no one with this eruption of the skin, while among my other patients it is quite frequent, especially among young chlorotic women. In them the physical signs of phthisis are indefinite, but amenorrhea is frequent. In advanced cases we often meet with brownish coloration of the skin, mostly marked on the face, but at times all over the body, simulating the smoky gray or bronze color seen in Addison's disease. Considering the frequency with which the adrenals are found affected in consumptives, we have an explanation for this phenomenon.

Patients who sweat profusely may show *miliaria*, or *sudamina*, on the chest and abdomen. *Herpes zoster* of the trunk and limbs may also occur, mostly in patients with caries of the spine.

Pityriasis Tabescentium.—In more or less advanced cases other skin eruptions are often seen which are, within certain limits, characteristic of phthisis. In those who sweat profusely the atrophied skin is during the day dry, pale, and brittle, and the upper epidermic layer desquamates and sheds yellow or gray scales. In some cases it looks as if the skin was covered with dust. It is known as pityriasis tabescentium and occurs mostly in consumptives who are not extremely emaciated, but who have excessive secretion of sweat and sebum; it is localized over the chest anteriorly and posteriorly, but at times the entire body is covered with it. It may be seen in other wasting diseases, but most often in phthisis.

Pityriasis Versicolor.—This is even more often seen in phthisis. The eruption is discretely scattered over the anterior and posterior aspects of the thorax, and consists of small macules, slightly raised above the level of the skin, round or oval in shape with well-defined margins. Scales can be scratched off and when examined show roundish, shining microscopic spores, the *Microsporon furfur*.

FIG. 47.—Papulonecrotic tuberculides. In this patient the eruption occurred on the extremities (Darier.)

The color of the eruption varies in different individuals, but is mostly brown, or a dirty yellow, darker in those who lead an outdoor life, and over the arms and neck when these are affected, while in negroes they are almost white. In patients who neglect to attend to the cleanliness of their bodies the macules may coalesce, forming large, irregular plaques covering large tracts of skin anteriorly and posteriorly, which desquamate upon scratching.

It is seen in consumptives who sweat profusely at night, which favors the growth of the fungi, and in patients whose skin has a tendency to scale, which assists in their detachment. Piéry[1] has inoculated guinea-pigs with the scales removed from such patients and obtained positive results, and he suggests that it is a tuberculous dermatomycosis.

When seen on the chest, pityriasis versicolor is fairly indicative of phthisis, although it occurs in other cachectic diseases, notably cancer.

[1] Gaz. de hôp., 1912, 85, 531.

Tuberculides.—This term was suggested in 1896 by Darier to designate certain skin eruptions which are found in connection with tuberculous infection and disease. Of the various types which have been classified under this term, only *acnitis* and *folliclis* are of special interest to those who care for tuberculous patients.

Acnitis is seen most in adolescent or young adult individuals and is found mainly on the face, rarely on the limbs, and genital organs. The eruption consists of either small, red, or dark brown, circumscribed, and indolent nodules or papules. Each lesion lasts about a month when it is either absorbed or, more commonly, it suppurates and opens on

FIG. 48.—Acnitis in a man aged twenty years. (Darier.)

the surface leaving a small scar behind. Crops appear one after another so that we can always see several of these papules in various stages of development symmetrically scattered over the face, and at times over the back between the shoulder blades. It is at times difficult to differentiate these papules from acne, but the absence of seborrhea and comedones favors the diagnosis of acnitis.

Folliclis is also seen in youthful persons, the eruption appearing in successive crops on the forearms, hand, fingers, elbows, knees, feet, and ears, and rarely on the face. It is not rare on the trunk, especially over the shoulder-blades. The papule is dark red, which is within about a week transformed into a pustule at the summit. The pustule

dries, and finally crusts, and this is shed in a couple of weeks, leaving a depressed scar. In nearly all cases we meet with crops in various stages of development.

There seems to be no question that these eruptions are etiologically associated with tuberculous infection. But they are mainly associated with glandular, osseous, serous, visceral or cutaneous tuberculosis. It is also noteworthy that they are mainly found in cases in which the just-mentioned tuberculous lesions are either latent, or quiescent, and in the numerous cases of pulmonary tuberculosis in which the writer has observed folliclis and acnitis, the lung lesions were inactive. Many dermatologists, notably Darier,[1] Jadassohn,[2] Lewandowsky,[3] Stokes,[4] and others, point out that tubercle bacilli are only rarely found in sections of the papules, or in the pus, and even inoculation experiments prove negative in most cases. The histological structure has been found typically tuberculous only in exceptional cases; in most cases it is found inflammatory and necrotic. However, it is the consensus of opinion that they are due to emboli of tubercle bacilli circulating in the blood and which are deposited in the hypersensitized skin, calling forth an allergic response.

Elsewhere in this book (see Chapter XXX) we have shown that there appears to be a decided antagonism between tuberculous lesions of the skin and the lungs. These dermic lesions seem to confirm this observation. All writers, notably Darier, Lewandowsky, and Stokes, point out that while most patients have either a family history of tuberculosis, or symptoms of latent tuberculous disease of the lungs, gland or bones, the eruption itself runs an afebrile course and constitutional symptoms are almost invariably lacking. It is therefore advisable to reassure the patient that while cosmetically the disease is a nuisance, there is little danger of developing active pulmonary tuberculosis. In patients with confirmed phthisis the appearance of acnitis or folliclis is an indication of a good prognosis as regards the lung lesion.

The Hair.—Many authors have stated that alopecia is more frequent in phthisical subjects than in others, and it has been attributed to the same causes as those operative when the hair falls out after an attack of typhoid fever, etc. But in my experience this is not true. The tuberculous patients in my hospital and private practice are not more often bald than others of the same class, nor do I meet with many consumptives who have localized alopecia, or alopecia areata. Premature grayness of the hair, which Cornet mentions as very frequent among consumptives, has also not been found by me to be frequent in tuberculous patients in the United States.

Clubbed Fingers.—Clubbed fingers were already mentioned by Hippocrates as a symptom of phthisis, and French writers at present

[1] Dermatology, Philadelphia, 1920, p. 563.
[2] Tuberkulose der Haut, Berlin, 1906.
[3] Die Tuberkulose der Haut, Berlin, 1916.
[4] Am. Jour. Med. Sci., 1919, **157**, 313, 522.

call them *doigts hippocratiques*. They are found in about one-third of advanced consumptives, and are probably caused by chronic peripheral passive congestion. Clubbed fingers are not exclusively met with in phthisis, but also in empyema, bronchiectasis, chronic bronchitis, asthma, and pulmonary emphysema, in thoracic aneurysms, etc. They have also been encountered in rare cases of cirrhosis of the liver, cancer of the lung and amyloid disease.

In phthisis we usually find that the fingers of both hands are thickened and bulbous, like a club or drumstick, resembling somewhat the condition seen in chronic onychia. The terminal phalanges are enlarged, the nails curved longitudinally and laterally. In a recent study Corper, Cosman, Gilmore and Black,[1] refer to these changes as "the hyperconvex nail," and they state that it occurred to the extent of about 75 to 95 per cent of cases of pulmonary tuberculosis, there being no difference in the incidence dependent upon the activity of the disease or the sex of the case. In the writer's experience, the percentage of incidence has been much lower. From roentgenographic studies it appears that the bones and joints are not affected, nor is the skin altered in any way, but only the superficial soft parts are hypertrophied. As to what the change consists in we are in ignorance because of lack of anatomical and histological studies. Some have suggested that it is a fibrous thickening of the innermost layers of the epidermis, as a result of prolonged congestion of the capillaries. This may be true of some cases, but in those in which the condition develops within a few weeks it is doubtful whether this could be the actual anatomical change. Corper and his co-workers have found that hypertrophy of the soft tissues of the hands and feet of consumptives has a higher incidence in the active (about 65 to 80 per cent) than in inactive cases (about 45 per cent).

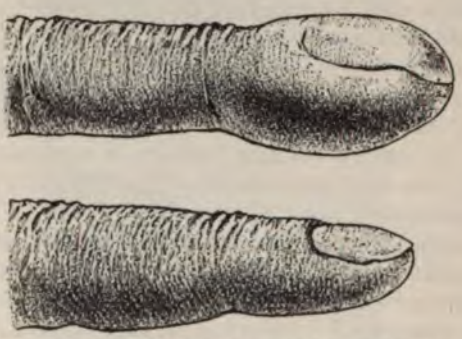


FIG. 49.—"Drumstick" fingers and curved nails.

In most cases the onset is slow and insidious and the patient knows nothing about it until the physician calls his attention to the clubbed

[1] Am. Rev. Tuberc., 1921, **5**, 357.

fingers. But in rare instances, as has already been noted by Trousseau, it comes on very quickly and within a few weeks the fingers look

FIG. 50.—Clubbed fingers in phthisis.

like drumsticks. In these acute cases they may be painful, tender, and livid. Lividity is also seen in those suffering from pulmonary

FIG. 51.—Changes in the toes in tuberculous osteo-arthropathy.

emphysema or fibroid phthisis. The nails are curved and look like claws.

My observations are in agreement with those of Bezançon[1] that clubbed fingers are not met with in all cases of chronic phthisis, as some have stated. A large number of consumptives have normal-shaped fingers, while some have even long, tapering terminal phalanges. Clubbed fingers are encountered almost exclusively in fibroid phthisis, pulmonary emphysema with tuberculosis and in those having extensive pleural adhesions. In other words, whenever clubbed fingers are encountered in a case of phthisis we find that the patient is also

FIG. 52.—Roentgenogram of a hand in a case of clubbed fingers in pulmonary osteo-arthropathy with bronchiectasis and pulmonary emphysema. On the tips of the end phalanges marked cauliflower formations; bony excrescences on basal portion of some phalanges; typical Heberden's nodes; broadening of the bases of the middle phalanges.

suffering from dyspnea and dilatation of the right heart. This would suggest mechanical disturbances of the circulation, causing peripheral venous stasis. Moreover, the prognosis in these cases is quite favorable as regards duration of life, though the outlook as to comfort is rather gloomy.

Hypertrophic Pulmonary Osteo-arthropathy.—In some cases of far-advanced tuberculous disease we meet with enlarged hands and feet simulating those of acromegaly. These changes in the extremities were

[1] Arch. gén. de méd., 1904, **1**, 1663; **2**, 3100.

first described by Pierre Marie,[1] and independently by Bamberger.[2] The fingers are altogether increased in volume, the nails enlarged, and curved transversely and longitudinally, like the beak of a parrot. In most cases the metacarpophalangeal region remains normal, but the wrist becomes enlarged and deformed, bulging on its dorsal aspect. In many cases there is also some deformity of the spine—kyphoscoliosis, and the feet may show changes similar to those observed in the wrists and hands, especially the toes and tarsus. In the cases that came

FIG. 53.—Roentgenograms of hand in a case of fibroid phthisis.

under the writer's observation there were pains of variable severity, in rare instances almost unbearable, and generally intermittent.

Milder forms of osteo-arthropathic changes are very common in tuberculous patients. From a recent study of these changes by Corper and co-workers, it appears that the gums are in the majority of cases anemic and hypertrophied, and bony processes decidedly thickened, more so in the superior maxilla than in the lower jaw. In about 25 per cent of cases of pulmonary tuberculosis Corper found that the hard palate reveals a thickening located in the midline and extending

[1] Rev. de Médecine, 1890, **10**, 1.
[2] Wien. klin. Wchnschr., 1889, **2**, 226; Ztschr. f. klin. Med., 1891, **18**, 193.

from the horizontal plate of the palate bone to the foramen of Scarpa, situated in the anterior portion of the palatal process of the superior maxilla, especially in pronounced cases. The tumor is distinctly hard and bone-like to the touch. It is Corper's opinion that these changes in the palate are part and parcel of the general hypertrophic osteo-arthropathy, and it is more common in active than in arrested cases. This swelling of the hard palate, known as *torus palatinus*, is, however, not found in tuberculous patients more often than in healthy individuals. An investigation by Herman Schwatt in my clinic at the Montefiore Hospital and the Bedford Sanatorium showed that it occurs in about 16 per cent of persons, tuberculous and others, and that neither tuberculosis, nor osteo-arthropathy, is a factor in its production.

We are in the dark as to the cause of these changes in the bones and joints in chronic pulmonary tuberculosis. As can be seen from the roentgenograms (Figs. 52 and 53) clubbed fingers and osteo-arthropathy differ in the fact that in the former only the soft parts are implicated, the bones remaining practically normal, while in the latter the tissues of the bones and joints are hypertrophied and some osteophytes may be seen at the line of the joint cartilages. From the studies of Leo Kessel,[1] Edwin A. Locke,[2] Montgomery,[3] Corper,[4] and others, clubbed fingers are the same process as osteo-arthropathy, and merely representing an early stage of it. Locke found early proliferative changes in the periosteum of the long bones of the forearm and lower legs in cases with clubbed fingers of exactly the same type as in hypertrophic osteo-arthropathy. Clinically we differentiate these two conditions by the fact that in clubbed fingers only the terminal phalanges are enlarged while in osteo-arthropathy the wrist is also affected, and the feet usually show the same changes, and in addition there is in most cases decided spinal deformity. But this does not exclude the identity of the two processes if we choose to regard clubbed fingers as the early stage of osteo-arthropathy. The former are, however, more common.

It is important to emphasize that these changes in the extremities are found, as a rule, in extremely chronic cases of tuberculosis, especially fibroid phthisis. In many, though the pulmonary lesion is undoubtedly active, it pursues a slow, sluggish course, the patient lasting for many years, and is able to perform light work. This is to be considered in connection with the benignity of phthisis in arthritics of other sorts, and in those in whom fibrosis is easily formed. This point is discussed in detail in Chapter XXX.

[1] Arch. Intern. Med., 1917, **19**, 239.
[2] Ibid., 1915, **15**, 659.
[3] Jour. Cutan. Dis., 1916, **34**, 285.
[4] Loc. cit.

CHAPTER XII.

SYMPTOMS REFERABLE TO THE CARDIOVASCULAR AND URINARY SYSTEMS.

THE CARDIOVASCULAR SYSTEM.

Cardiac Palpitation.—Of the functional cardiovascular disturbances in phthisis the most important are palpitation, tachycardia, and hypotension. They are very often associated, but at times we meet one to the exclusion of the other.

In incipient cases palpitation is mainly met with in young persons, especially chlorotic girls. Slight or moderate exertion, excitement, and emotional disturbances may cause an attack, or it may occur without any obvious provocation. At times it is very pronounced, and is perhaps the only subjective symptom which induced the patient to consult a physician. Rarely it is very severe and is accompanied by precordial pains and distress and by vasomotor disturbances, such as pallor, or flushing of the face, sweating, etc.

I have met with cases in which palpitation preceded all subjective and objective symptoms of incipient phthisis. Some are for this reason treated for heart disease. As will be shown when speaking of the differential diagnosis of phthisis, the syndrome known as hyperthyroidism is often mistaken for tuberculosis. The reverse is also true: Very frequently the rapid pulse, the tendency to sweating and flushes, emaciation, etc., are erroneously considered symptoms of hyperfunction of the thyroid, or neurocirculatory asthenia, and treated as such. A careful examination of the chest, however, will reveal a tuberculous lesion.

In some instances, especially in young persons, and in women during the menopause, symptoms of the *hypoasphyxial syndrome*, first described by Martinet,[1] clearly point to urgency to examine the lungs carefully. These individuals have more or less cyanosed facies, cold and cyanosed extremities, small pulse, vascular hypotension, peripheral venous stasis, and venous distention of the visceral organs shown by disfunction of the digestive and internal secretory glands. It is this class of tuberculous patients who are frequently treated for neurasthenia, hyperthyroidism, neurocirculatory asthenia, etc. On the other hand, experience has shown that, as a rule, while these functional symptoms may be very severe, the tuberculous lesions in the lungs are usually very mild showing slight, if any, tendency to progression.

[1] Diagnostic Clinique, Paris, 1919, p. 744.

In all probabilities this is due to the hyperfunction of the thyroid which, as will be shown later on (see Chapter XXX), exerts a favorable influence on the course of pulmonary tuberculosis.

The causes of the palpitation at this stage are not clear. Some have been inclined to attribute it to dilatation of the right heart, but we meet it in cases in which this organ is normal. Others believe it is due to the anemia—low arterial tension—or to sympathetic nerve disturbances. The last factor is apparently operative in many cases, because we meet it mostly in nervous patients, in young girls and in women during the menopause. Compression of the vagus by enlarged glands may be the cause in some cases. Of course, the toxemia of phthisis exerts a great influence on the heart, inducing fatty changes with resulting tachycardia and palpitation.

Cardiac irritability is seen also in advanced, but quiescent, cases. The patient is doing well, has no fever, no cough, and is not emaciated. But the least exertion, emotion, or complication, provokes cardiac distress which may be very painful, almost anginal. Here, the palpitation is, as a rule, due to cardiac dislocation, and occurs more often in left-sided lesions. A large cavity in the left lung with pulmonary contraction has drawn the mediastinum to the left, and the diaphragm upward, so that the heart is pushed upward and to the left, and the apex beat may be found in the third interspace at the axillary line. In some cases of this class there may also be arrhythmia. The palpitation is not so pronounced in right-sided dislocations of the heart, not even in complete dextrocardia.

Palpitation has no influence on the course of phthisis, excepting in the advanced stages when it is due to dislocation of the heart. In the early cases we may meet with annoying palpitation in nervous patients who are progressively improving. But from the diagnostic standpoint it is a symptom of great value. Hirtz said that "when a patient complains of palpitation, examine his lungs; and examine his heart when he complains of dyspnea." While this does not hold good in every case, yet it is well worth bearing in mind, especially when dealing with anemic youths. In some cases of phthisis we meet with palpitation for a day or two before the occurrence of hemoptysis.

Tachycardia.—*Rapid heart action objectively ascertained—which may not be known to the patient at all, thus differing from palpitation, which is a subjective symptom—is very frequent in all stages of phthisis.* In my experience, over 90 per cent of cases of incipient phthisis have tachycardia which is usually permanent or, rarely, paroxysmal. It is a symptom of phthisis which is not appreciated to the extent it deserves, though it is often very helpful in deciding a doubtful case.

The tachycardia may be of toxic origin. Every elevation of temperature in phthisis, as in other acute febrile conditions, is accompanied by an acceleration in the pulse-rate. But it is often pronounced in those running a subfebrile temperature and also in afebrile cases. In fact, *in tuberculosis the pulse is accelerated far out of proportion to the height*

of the temperature. In most other cases an elevation of 1° F. is usually accompanied by an increase in the pulse-rate of about eight beats per minute, while in phthisis we often have a temperature of 100° while the pulse counts 120 and even more. In fact, in most afebrile cases of phthisis the pulse is over 90 per minute and during the morning subnormal temperature tachycardia is not at all rare. Thus tachycardia is an early symptom of phthisis and some writers consider it a premonitory symptom.

Permanent Tachycardia.—In a large proportion of cases the tachycardia is permanent and accompanied by subjective discomfort, such as palpitation, languor, debility, dyspnea, etc. In others, it is purely objective; the patient is hardly aware of its presence. I have observed many cases in which the disease was arrested, or even cured, yet the tachycardia remained. At times it greatly interferes with the patient's efficiency. But I cannot agree with those who say that in an arrested case one cannot feel safe as to the continued favorable progress of the patient so long as the pulse-rate remains high. I have seen patients who have been able to work for a living without much discomfort in spite of the rapid heart action.

One characteristic of the pulse of the consumptive is its instability and variability. While resting the rate may be normal, but the slightest exertion—a fit of coughing, some emotional experience, a heavy meal, or changing from the reclining to the erect posture—may send up the pulse-rate to 110 or 120. Faisans maintained that he did not know of any disease in which the pulse is as unstable as in phthisis.

Paroxysmal Tachycardia.—In rare cases we meet with paroxysmal tachycardia. The patient feels comparatively well, and, without any exciting cause, he is seized with severe palpitation, dyspnea, or even orthopnea and cyanosis. Counting the pulse-rate, we find it 150 to 200 per minute, small, wiry and often irregular. The attack may last a few hours, a day or two. In one case the patient got an attack while in my office, the pulse going up from 96 to 160, and looked as if he was breathing his last. He recovered in two hours. There was in my wards at the Montefiore Hospital a young woman who often got these attacks. In the beginning the rapid pulse, dyspnea, cyanosis, and prostration suggested the collapse characteristic of pneumothorax. Careful search for signs of air in the pleura proved negative. She got these attacks at irregular intervals and recovered within a few hours or a day.

After several attacks, which may come on at frequent intervals, we may observe signs of cardiac dilatation—the heart gives way and the result is edema of the lower extremities, enlargement of the liver, etc. Finally, asystole occurs and the patient succumbs. Paroxysmal tachycardia is of grave significance and, when occurring several times, will ultimately kill the patient during one of the attacks. Instances of sudden death are not unknown among tuberculous subjects. The cause is frequently heart failure.

Causes of Tachycardia.—The causes are obscure. It has been attributed to bulbar lesions, to interstitial neuritis of the pneumogastric nerve, to myocarditis, etc. Some believe that it is due to compression of the vagus by enlarged tracheobronchial glands, but it would seem that the effect should rather be a slowing of the pulse-rate, than an acceleration. Indeed, considering that the vagus is often pressed upon by enlarged glands, it is noteworthy that a slow pulse is exceedingly rare in phthisis. Other authors have attempted to explain this phenomenon by stating that it depends on which part or branch of the pneumogastric is affected by the tuberculous process. On this also depends whether the stomach or myocardium will suffer. K. Bohland[1] is inclined to ascribe the tachycardia in phthisis to the small heart characteristic of the disease—in order to pump enough blood into the system, the heart must beat more often. In the advanced stages of phthisis it is due to myocarditis. The tuberculous toxemia alone does not explain the tachycardia because it is found often in afebrile patients, as was already stated.

Permanent tachycardia aggravates the prognosis of phthisis, and these patients should not be sent to a high altitude. The causes are complex and vary with each case. In patients in whom it is of toxic origin we may expect improvement as soon as the fever subsides. But in many it is caused by compression of the pneumogastric nerve by enlarged tracheobronchial glands, or an adherent apical pleura, neuritis of that nerve, or reflexly of gastric origin, fibrous degeneration of the cardiac muscle, or tuberculosis or hyperfunction of the adrenals, etc. When due to cardiac displacement, especially to the left in left-sided lesions, it is permanent.

Arrhythmia is only rarely observed in phthisis, and the prognosis of these cases is rather unfavorable.

Bradycardia.—*A slow pulse is exceedingly rare in phthisis;* those who see large numbers of these patients occasionally meet one with a pulse less than 50 per minute. One patient under my care had a pulse-rate of 36 per minute for several months, and only during febrile attacks did it rise to 50 or slightly more. But here there was probably an organic heart-block. Gueneau de Mussy, who described some of these cases, attributed it to irritation of the pneumogastric nerve. On the other hand, there are many physicians of large experience who have never seen bradycardia in phthisis. From the few cases met by me, it appears that the prognosis in phthisis with a slow pulse is very good.

At the terminal stage of far-advanced phthisis we often meet with a slow, soft, almost imperceptible pulse which intermits, indicating cardiac failure, or exhaustion. The pulse is also slowed when meningeal irritation complicates the disease.

[1] In Brauer, Schröder and Blumenfeld's Handbuch der Tuberkulose, 1915, **4**, 4.

Arterial Hypotension.—The blood-pressure, measured with a sphygmomanometer, is lower than normal in the vast majority of phthisical patients. It is evidently due to the toxic effects of the metabolic processes of the tubercle bacilli, because an injection of tuberculin is usually followed by a decided fall in the blood-pressure. Sir Douglas Powell says that the large doses of tuberculin which were used in the first days of Koch treatment of lupoid and other forms of tuberculosis caused severe collapse, and recent writers, like Levy, Geisbock, and others, found that even in small or moderate doses, tuberculin reduces arterial tension. It has been found that a low blood-pressure is an almost constant characteristic of the very early stages of phthisis and, when occurring in an adult without any other assignable cause, tuberculosis is to be suspected. My own experience has brought me to the conclusion that in cases presenting obscure symptoms and signs of phthisis, when accompanied by a low blood-pressure, the diagnosis may be safely made; conversely, I always hesitate in cases with high arterial tension, excepting in persons over fifty years of age. But even in these high pressure is rare in phthisis.

This hypotension is quite marked in the early stages and becomes more accentuated with the progress of the disease. I find that, as a rule, *cases of undoubted phthisis with a normal or high blood-pressure have a favorable prognosis.* This is the case with phthisis in persons having interstitial nephritis, gout, pulmonary emphysema, syphilitic endarteritis, etc.; they all have high blood-pressure, and the prognosis is favorable as regards the tuberculous disease. *When the blood-pressure is low at first but rises gradually, it is an excellent indication of improvement;* conversely, tuberculous patients with normal or high blood-pressure who begin to show hypotension almost invariably also show indications of the extension of the process in the lung and the prognosis is aggravated. I have not noted in many cases any relation between the hypotension of phthisis and the temperature, the pulse-rate, or the dyspnea. It is met with in febrile and afebrile cases; in young and in the aged.

It has also been observed by many authors that patients with a tendency to hemoptysis have a high blood-pressure which rises before the onset of the bleeding. At one time I tested this point in several patients but could not confirm it. Many who bled profusely had a very low blood-pressure.

THE BLOOD.

The Erythrocytes.—Despite the external appearance of anemia frequently seen in many phthisical patients in all stages of the disease —which has given rise to the expression "great white plague"—no changes in the cytology of the blood characteristic of the disease have been found. In fact, it is noteworthy that many patients who look pale show an almost normal blood picture. Only in the advanced stages, as a rule, is there to be noted a decrease in the percentage of

hemoglobin. In many cases, even with high fever, the hemoglobin contents remain normal. At times a polycythemia is encountered, but the hemoglobin is not increased under the circumstances. In rare instances I have found a decided decrease in the number of erythrocytes, especially during the very early, and very advanced, stages of the disease. In some few cases the count was as low as 1,000,000, or even less, but the fact that it is so rare shows that it is an accidental occurrence, and cannot be considered characteristic of the disease. After profuse pulmonary hemorrhages the anemia may be profound, but it is remarkable that the blood picture improves very rapidly after the cessation of bleeding.

There is at times noted a decidedly low percentage of hemoglobin in incipient cases, even when the erythrocytes are not decreased in number. For this reason some authors have spoken of a pseudochlorotic blood picture. But soon after the patient is placed under proper dietetic and hygienic treatment the hemoglobin content of the blood improves, as a rule. It may be stated that in many cases there is slight diminution in the number of erythrocytes, and a pronounced diminution in the hemoglobin content, during the incipient and far-advanced stages of phthisis.

From the researches of Limbeck, Grawitz, and others it appears that with the advance of the disease, even with the formation of pulmonary excavations, the blood picture is very often not deviating from the normal. The yellowish pallor, "ochrodermia," which is so frequent at this stage, is not due to alterations in the cytology of the blood, so far as can be ascertained. But there is good reason to believe that the total amount of blood in the body is less than in healthy individuals; that there is a distinct oligemia. This has been ascribed to the loss of water through profuse nightsweats, expectoration, and often diarrhea, which brings about a higher specific gravity of the blood with a concentration of the cells.

In the far-advanced stages, with hectic fever, often complicated by mixed infection, there is, in addition to leukocytosis, a diminution in the number of erythrocytes, with a fall in the percentage of hemoglobin.

Leukocytes.—In incipient phthisis the leukocytes are quite normal in number and variety. Even in acute cases, so long as there is no mixed infection, the leukocyte count is unaffected. Some authors, notably Ullom and Craig[1] in this country, have found a slight leukocytosis which increases somewhat with the advance of the disease. But inasmuch as it only reaches about 11,000 to 14,000 on the average, it cannot be considered of any value diagnostically. We quite often find that in women the number of white-blood cells oscillates between 4000 and 15,000 under normal physiological conditions.

Accumulated evidence is in agreement with the findings of Blumen-

[1] Am. Jour. Med. Sci., 1905, **130**, 386.

feldt[1] to the effect that the total number of leukocytes is somewhat higher in the tuberculous than in healthy persons. While an absolute leukocytosis may be found in the early stages of the disease, a relative leukocytosis is only found in the advanced stages, and it apparently runs parallel with the gravity of the tuberculous lesion in the lungs. Eosinophiles are diminished in number relatively and absolutely in the advanced stages. But even this is an inconstant phenomenon. It may be stated that the tubercle bacillus, or its toxin, exerts no specific positive chemotaxic influence on the leukocytes, and we are not justified in ascribing a typical influence and affinity of the tubercle bacilli on the two main varieties of white cells in the blood.

Gerald B. Webb, G. B. Gilbert, and L. C. Haven[2] found the blood platelets are increased in number in cases of phthisis. In tuberculosis in guinea-pigs they observed the same phenomenon. They believe that the blood platelets either contain or supply opsonins. The fact that they are increased at an altitude of 6000 feet would, according to Webb, point to a reason for the salutary effects of high climates on phthisical patients.

With the advance of the disease leukocytosis is not rare; it is usually transient, but rarely permanent. It appears to depend on the activity of the tuberculous process, the intensity of the fever, the presence of complications, etc. But there are so many exceptions to this rule that it cannot be utilized for diagnostic and prognostic purposes. It appears, however, that an injection of tuberculin is usually followed by transient leukocytosis. Some have attempted to judge the presence of excavation by the white-cell picture, but have failed. Wright's attempt to utilize his tuberculo-opsonic index in the prognosis of tuberculosis has also failed to give satisfaction to most authors.

Arneth's Blood Picture.—A great deal has been made by some authors of Arneth's blood work in infectious diseases, especially tuberculosis. His theory is based on his observations of the growth of the neutrophile and the changes of the nuclei, or granules within these cells during the period. He developed a very complicated blood picture, based on the number of granules or fragments in each neutrophile. His contention is that when the disease takes a bad turn, there is an increase in the number of young forms of neutrophiles containing but one granule as a nucleus, and a decrease in the older forms of cells which correspond to the polymorphonuclears of other writers; he calls it a shifting of the blood picture to the left.

This method has been given a trial by many authors and they, as a rule, could find no diagnostic or prognostic hints which were constant; in fact, the contradictions were so frequent and notorious that it has been abandoned altogether by nearly all who were originally enthusiastic in its favor.

[1] Ztschr. f. exper. Pathol. u. Therap., 1919, **20**, 14.
[2] Arch. Int. Med., 1914, **14**, 743.

Tubercle Bacilli in the Circulating Blood.—That the virus of tuberculosis may be found in the blood has been suspected for a long time. As far back as 1866, Villemin withdrew blood from the femoral artery of a tuberculous rabbit, and also used blood drawn by leeches from a phthisical patient, and injected it into a guinea-pig, and thus transmitted the disease. Marcet[1] repeated these experiments in 1867. Weichselbaum, in 1884, found tubercle bacilli in the blood of patients suffering from miliary tuberculosis, and more recently many authors have reported positive findings in patients with chronic, or even quiescent, tuberculous lesions. Liebermeister[2] found them in 75 per cent of cases a week or two before death, and in many early cases of tuberculosis. Some authors, notably Rosenberger,[3] Koslow, Kurashige,[4] etc., have even stated that in all cases of tuberculosis bacilli may be found when carefully looked for, while Klemperer found them in 7 cases in which the disease was only suspected but could not be diagnosticated with the usual clinical methods.[5]

At one time great enthusiasm was entertained that here we have a reliable method for the recognition of obscure tuberculous disease by simply ascertaining a bacteremia before the bacilli have had an opportunity to settle in some organ and produce gross pathological changes perceptible to the clinician. But then still other investigators, like Liebermeister, Suzuki and Takaki,[6] and Kurashige, discovered tubercle bacilli in the blood of apparently healthy individuals, and Clara Kennerknecht,[7] in the blood of 91 per cent of 120 apparently healthy children, of which only 68 were tuberculous on subsequent observation, and the hopes entertained for the clinical application of this method of diagnosis vanished.

Further investigations by Walter V. Brehm,[8] Beitzke, Schern, and Dold have shown that there was a source of error which was not considered by most of these bacteriologists. The tap water used in diluting the blood often contains acid-fast rods which look like tubercle bacilli under the microscope (see p. 30). These acid-alcohol-fast rods may be bacilli, or some other substances, but they are not pathogenic to susceptible animals. It has also been found that fragments of red-blood corpuscles may take on the stain of the tubercle bacilli and show acid-fast properties.

The microscopic test proving unreliable, inoculation experiments have been made to clear up the problem. The blood of tuberculous patients was injected into susceptible animals. Some authors, like

[1] Quoted from Calmette, L'infection Bacillaire, p. 216.
[2] Virch. Arch., 1909, **197**, 332; Med. Klinik, 1912, p. 798.
[3] Am. Jour. Med. Sci., 1909, **137**, 267.
[4] Ztschr. f. Tuberkul., 1911, **17**, 347; 1912, **18**, 430.
[5] For a résumé of the literature on this subject, see, Löwenstein, Vorlesungen über Tuberkulose, Jena, 1920, pp. 146–164.
[6] Zentralbl. f. Bakteriol., 1910, **61**, 149.
[7] Beitr. z. Klinik. d. Tuberkul., 1912, **23**, 265.
[8] Jour. Am. Med. Assn., 1909, **53**, 909.

Anderson,[1] Rumpf,[2] Ravenel and Smith,[3] Querner,[4] Leo Kessel,[5] and others, were either entirely negative, none of the animals experimented on showed any tuberculous lesions after death, or only a few of them were infected. Liebermeister, on the other hand, reported that in 6 cases the animals were infected with tubercle bacilli in the blood of human beings who showed no clinical symptoms of the disease.

Investigations by Mildred C. Clough[6] showed that inoculation tests are unreliable, especially when the blood is drawn from patients suffering from chronic phthisis. She collected from the literature 1508 cases studied by guinea-pig inoculation, of which 195, or 12.9 per cent, gave positive results. In 500 cases Fränkel[7] found 20 per cent positives; Fischer[8] in 1250 cases, 17 per cent. However, in all these cases acute and chronic tuberculosis were indiscriminately mixed together. In 48 cases of miliary tuberculosis, 66.6 per cent gave positive results. In other words, according to Clough, only 6.7 per cent of chronic cases, and 66.7 per cent of acute cases give positive results to inoculation tests. Miss Clough says that with blood cultures positive results are more often obtained of the existence of a bacteremia in tuberculosis.

The reason why tubercle bacilli in the circulating blood are only rarely pathogenic to susceptible animals has been given differently by different authors. Some maintain that it all depends on the number of bacilli in the injected blood, and when they are scarce, they may prove ineffective. Moreover, while inoculating the blood not only bacilli are injected, but also antibodies, and that the bacilli, while remaining in the living blood, have been attenuated in their virulence. If their virulence had been maintained, acute miliary tuberculosis would have resulted in the patient from whom the blood was drawn.

From the theoretical viewpoint it is important to mention that it is agreed that while circulating in the living blood tubercle bacilli do not multiply by proliferation, it is only when they settle on some susceptible tissue that they may grow in number through multiplication.

So far, the finding of tubercle bacilli in the blood is of no diagnostic or prognostic value, even in suspected cases of acute miliary tuberculosis.

THE URINARY SYSTEM.

The Kidneys.—There appear to be no changes in the structure and functions of the kidneys which can be considered specific and characteristic of early phthisis, excepting in cases with a very acute onset,

[1] The Presence of Tubercle Bacilli in the Blood in Clinical and Experimental Tuberculosis, Hygienic Laborat. Bull., Washington, 1909, No. 57.
[2] München. med. Wchnschr., 1912, **59**, 1951.
[3] Jour. Am. Med. Assn., 1909, **53**, 1915.
[4] München. med. Wchnschr., 1913, **60**, 401.
[5] Am. Jour. Med. Sci., 1915, **150**, 337.
[6] Am. Rev. of Tubercul., 1917, **1**, 598.
[7] Schmidt's Jahrbücher, 1913, **217**, 2056.
[8] Ztschr. f. Hygiene, 1914, **78**, 253.

with high fever, which affects these organs in the same manner as hyperthermia due to other causes, or in cases in which the kidneys are inoculated at the onset together with many other organs, as in acute miliary tuberculosis. Recent investigations of the renal function by Charles W. Mills[1] and John T. Henderson, and also by Elmer H. Funk,[2] show that in the incipient stage it is normal, and that it is reduced in the advanced stages of the disease only when there is evidence of structural damage to the kidney.

Some writers, notably the French, have described polyuria, phosphaturia and albuminuria as very frequent in early and even in latent phthisis. Barbier[3] says that albuminuria is often the only sign observed for a long time before other symptoms make their appearance; and that this albuminuria is often misunderstood by physicians. Albert Robin[4] describes pretuberculous polyuria: The quantity of urine in the early stage is increased; in the second stage normal; and in the third stage diminished, although some patients have polyuria throughout the course of the disease. The oliguria of the advanced stage is closely related to the fever, sweats, and eventual diarrhea. Robin maintains that the polyuria of early phthisis is simple, showing no abnormal constituents, or, at most, there may be phosphaturia,when, at times, it may be severe enough to cause irritation of the kidney substance, congestion, and, finally, albuminuria.

These changes have, however, not been met with sufficient constancy to place them in the category of pathognomonic or specific symptoms of early tuberculosis. Among 100 cases of early tuberculosis that I have especially investigated for the purpose of testing this point, I found albuminuria in only 9 cases and casts in only 3.

Albuminuria in Advanced Cases.—In the advanced stages albuminuria is very frequent. Montgomery found albumin present in about one-third of cases of phthisis. In the majority of cases the amount was only a trace and when found in larger amounts it was always associated with casts and blood or pus. By using delicate methods Mills and Henderson found traces of albumin, with or without hyaline casts, in 40 per cent of sanatorium patients. It appears that cases with intestinal ulcers have larger amounts of albumin than others. From his studies Montgomery arrives at the conclusion that a large number of casts in the urine of consumptives are indicative of an unfavorable prognosis, and the reverse.

As to the causes of the albuminuria we are not clear. Some look upon it as caused by the irritation of the tuberculous toxins, which are eliminated with the urine, on the renal parenchyma, while others see in it the effects of the chronic fever, or actual tuberculosis of the kidneys. In an exhaustive study of the problem, N. Leon-Kindberg[5] arrives at

[1] Am. Rev. Tubere., 1917, **1**, 574.
[2] Ibid., 1918, **1**, 145.
[3] Brouardel and Gilbert's Traité de Médecine, Paris, 1910, **29**, 423.
[4] Traitement de la tuberculose, p. 498.
[5] Études sur le rein des tuberculeux, Paris, 1913.

the conclusion that the so-called "tuberculotoxins" cause no lesions in the kidneys. The presence of isolated tubercles in the kidneys explains perhaps some cases of bacteriuria.

It must be mentioned that mixed infection, such as is seen in pulmonary cavities containing, in addition to tubercle bacilli, also pyogenic microörganisms, is usually the cause of albuminuria in the advanced stages of phthisis where there is no concomitant renal tuberculosis.

Nephritis in the Course of Phthisis.—Symptoms of acute nephritis are very rarely met with during the course of phthisis; but the chronic forms, parenchymatous and interstitial, have, however, been found in variable proportions. Bamberger found nephritis together with phthisis to the extent of 15 per cent; Potain states that one-fifth of all consumptives have nephritis; and others have found even higher percentages. Senator was inclined to the opinion that tuberculosis is an important etiological factor in chronic parenchymatous nephritis. But it appears that clinical symptoms of nephritis are usually altogether absent, even when albumin and casts are found in the urine, and cardiac hypertrophy is exceedingly rare.

Most of these views are based on the presence of albumin in the urine, and Montgomery[1] has shown that in pulmonary tuberculosis albumin and casts are not often associated with evidences of nephritis. *In phthisis, albuminuria is not necessarily a manifestation of nephritis, or even of renal tuberculous lesions;* but in many cases, especially in fibroid phthisis and emphysema, it is due to cardiac dilatation, to intestinal and hepatic disturbances, etc., which are so frequent in advanced phthisis. Albuminuria may also be the sole indication of a tuberculous lesion in a kidney which manifests itself by no other symptom during life. Thus, in a painstaking study of 106 pairs of kidneys taken from consumptives, made by J. Walsh,[2] 53.9 per cent were found to contain tubercles. He also found that among these 106 pairs of kidneys only 10 showed chronic interstitial nephritis, while in 44 kidneys from patients suffering from other chronic diseases, there were 23 with this form of nephritis, which clearly indicates that tuberculosis of the lungs is antagonistic to the ordinary chronic general interstitial nephritis, just as it appears antagonistic to general sclerosis of other organs.

The Amyloid Kidney.—In the far-advanced stages of phthisis with large suppurating cavities in the lungs, we often encounter amyloid degeneration of the kidneys, as in suppuration due to other causes. It is usually found associated with amyloid changes in other organs, notably the liver, spleen, and intestines. But even this is not as frequent as would be expected. White found 9.2 per cent; Walsh 6.6 per cent, and he never found it exclusively in the kidneys; Blum in only 6 per cent, but he points out that 79.2 per cent of all amyloids were caused by tuberculosis, of which 54.4 per cent is pulmonary phthisis.

[1] Fourth Annual Report Henry Phipps Institute, 1908, p. 120.
[2] Tr. Sixth Intern. Congr. Tuberc., 1908, 1, 347.

Its symptomatology is that of amyloid disease of the liver and intestines, and because it is always associated with other changes in the kidneys, such as chronic parenchymatous nephritis, the resulting symptoms are always complex. Albumin is usually present in the urine. I find it safe to conclude, when the liver is enlarged and there is profuse diarrhea, that there is no doubt that the kidneys are amyloid. But when there is no diarrhea, there is polyuria of low specific gravity, casts, and but little albumin.

Terminal Edema.—Edema is present in a large proportion of cases of advanced phthisis; the ankles and knees especially are thus affected during the terminal stages, but it does not always depend on the condition of the kidneys. Montgomery found no relation between edema and the occurrence of albumin and casts in the urine, and suggests that the edema found in tuberculosis does not depend primarily on nephritis. General anasarca is at times seen in far-advanced cases toward the end, and this may be a manifestation of the state of the kidneys, but when we bear in mind that in these cases we also have cardiac dilatation, it is clear that the pathogenesis is often complex.

The edema may be considered an ill omen, and I have not seen a consumptive with edematous ankles and knees survive, or even improve. It may be unilateral, sometimes one-half of the body is swollen and pitting, corresponding to the side on which the patient lies. At times we see it only in one upper extremity, due to pressure on the veins coming to the arm by tuberculous glands, or when they are implicated in the adhesive pleurisy of that side, and more commonly by thrombosis of the innominate, subclavian, or other veins. Phlebitis or thrombosis of the femoral, popliteal, and crural veins is even more frequent (see Chapter XXIX).

Uremia.—Symptoms of uremia are not often met with in phthisis, but not so rarely as some authors would lead us to believe. In the advanced stages we meet at times with typical uremia, which is often mistaken for meningeal infection. I have seen several cases of convulsions due to this cause. In severe dyspnea without fever, arising suddenly, uremia is to be thought of in cases with albumin and casts in the urine. Often the diarrhea observed in these cases is distinctly of uremic origin, and at times we meet with pulmonary edema. These conditions are usually very difficult of recognition and differentiation.

CHAPTER XIII.

NERVOUS SYMPTOMS OF PHTHISIS.

As an exquisitely chronic disease, phthisis is accompanied by many morbid manifestations of the nervous system; in fact, nearly every symptom of the disease is often influenced by the effects of the tuberculous toxins on the nervous system. The neurotic phenomena may make their appearance immediately at the outset, in some they precede the actual onset of active phthisis, while most confirmed consumptives have a psychology peculiarly their own, and show symptoms of nervous aberration which cannot escape the vigilance of the observant physician.

Neurasthenia and Psychasthenia.—The onset of phthisis is often accompanied by symptoms simulating that syndrome which is known under the vague term of neurasthenia; indeed, many patients have been treated for neurasthenia for months before the true nature of their affection was recognized. These symptoms have been described by many authors and deserve careful consideration.

A large proportion of incipient and confirmed consumptives complain of vertigo, headache, pains along the spine, irritability of temper, insomnia, not necessarily due to nightsweats, and fleeting pains of the chest which, at times, cannot be attributed to circumscribed pleurisy; frequent attacks of tachycardia, irrespective of the temperature and cardiac palpitation, are not rare. There is also the characteristic languor and persistent weariness, which is not relieved by sleep; on the contrary, many state that they feel weary and tired in the morning, on getting out of bed, and that this tired feeling wears off in the afternoon or evening, all of which is suggestive of neurasthenia and psychasthenia. Considering these symptoms there is little wonder that many patients are treated for "nervousness" until an attack of dry or moist pleurisy, or of hemoptysis, or a careful examination of the chest, reveals the true state of affairs. Papillon[1] goes so far as to say that he suspects every victim of neurasthenia to be a subject of latent tuberculosis, and G. D. Head[2] believes that a considerable proportion of neurasthenics harbor a tuberculous infection which is so concealed that it escapes detection by the usual clinical methods. Considering that neurasthenia is quite often the result of toxic causes, it is clear that tuberculous toxemia may be a cause of these symptoms in many cases. If the chests of all patients treated for neurasthenia

[1] Arch. de Scien. Méd., 1900, **5**, 19.
[2] Jour. Am. Med. Assn., 1914, **63**, 996.

were carefully examined, a large proportion of phthisis which is now only recognized in the advanced stages would be identified at earlier stages.

Reflex Nervous Phenomena.—Aberrations of the sympathetic or parasympathetic nervous system are not rare in phthisis. Among these may be mentioned the unilateral flushes of the face and occasionally of one ear, combined with a feeling of warmth, sweating, etc. In some cases it has been observed that the cutaneous temperature is higher on one side of the chest. These unilateral symptoms are usually found on the side corresponding to the affected hemithorax, and in bilateral lesions, to the side in which the recent, or more active, lesion is located. In some patients with extensive excavations in the lung, the nostril corresponding to the affected side is widely dilated. Dermographism is very frequent.

These disturbances in the autonomic system have, during recent years, been studied by many authors. Meyer Solis-Cohen[1] looks upon them as a defense reaction of the internal secretory organs to the tuberculous toxemia. D'Oelsnitz and Cornil[2] attribute them to irritation of the cervical sympathetic and found that they can be best detected by the oscillometer. They suggest the oscillometer, applied to the arm corresponding to the affected side, as a useful diagnostic measure to supplement routine diagnostic methods for the detection of latent intrathoracic lesions. Ernst P. Boas,[3] who studied the problem with the Goetsch epinephrin test, arrives at the conclusion that the functional disturbances of the circulatory, alimentary, and nervous systems in the tuberculous are not specific manifestations of tuberculosis, but are usually conditioned by a constitutional nervous instability of the patient. At times they may appear without such an underlying predisposition, and then they may bear some relationship to hyperactivity of the thyroid. When latent, these vasomotor and other functional disturbances, may be activated by tuberculous infection, and they appear to belong to the same group as, and are analogous to, those of neurocirculatory asthenia.

An important symptom of phthisis is dilatation of the pupils, to which Rogue,[4] Destrée,[5] and also T. F. Harrington[6] drew attention. Harrington described the widely dilated pupils as "not a paralyzed pupil, but rather one which seems to be in a more or less constant state of dilatation, due to some irritation along the track of the nerve fibers in the celiospinal region," and says that they may be found in cases before the evidences of active disease can be discovered. Dilatation of but one pupil is more frequent, some authors saying that it occurs in more than 50 per cent of cases; that it is an early symptom and may

[1] Am. Rev. Tuberc., 1917, **1**, 289.
[2] Bull. de la Soc. des Hôp. de Paris, 1919, **43**, 861.
[3] Am. Rev. Tuberc., 1920, **4**, 455.
[4] Gaz. méd., de Paris, 1869.
[5] Jour. de méd. et de pharm., 1894, 241.
[6] Boston Med. and Surg. Jour., 1899, **151**, 575.

be found before other symptoms and signs make their appearance. More recently, Meyer Solis-Cohen, Emil Sergent,[1] and H. Saint-Aude[2] have given this symptom attention. Sergent has shown that this sign is not peculiar to syphilis, but that it is very frequently encountered in pleuropulmonary tuberculous lesions and especially in chronic phthisis. He distinguishes several varieties. The inequality may be an isolated phenomenon, and the abnormal pupil is on the same side as the affected lung. It may also be a part of an oculopupillary syndrome, myosis being accompanied by diminution of the palpebral fissure and retraction of the eyeball on the affected side. In some cases the inequality of the pupils not only forms part of the oculopupillary syndrome, but is also accompanied by vasomotor symptoms in the cheek and ear on the same side. Instead of myosis of the pupil on the side corresponding to the affected lung, there is mydriasis with vasomotor symptoms, but without the oculopupillary syndrome. It is mainly seen in cases of phthisis in which the apical pleura is involved in the process, and in apical pleurisy. In most cases of this type there is also found some swelling of the supraclavicular glands (see p. 486). It is due to irritation of the cervical sympathetic by the inflammatory process in the lung apex and pleura. With the improvement in the disease the difference in the pupils may disappear, but I have seen it persist after the patient recovered. At times, one pupil is unduly contracted.

Muralt[3] pointed out that these unilateral nervous phenomena may be observed within certain limits experimentally after the induction of therapeutic pneumothorax. He found that with the increase in the intrapleural pressure, the pupil dilates and the cheek flushes on the affected side, and in some cases there are typical attacks of migraine, while with the decrease in the pressure the phenomena disappear.

Pains.—While a large proportion of tuberculous patients pass through the disease painlessly, there are many who suffer from pains and aches of various degrees of severity. The pains may be in any part of the body, but the most characteristic are those of the chest and upper extremity. Kuthy found that among 650 patients, 60 per cent had pains in the chest, and of these it was localized in 85 per cent in the affected, or more affected, side.

Many of my patients have received the first intimation of trouble with their lungs through pains which were usually felt in the infraclavicular space above the second rib, and more often in the supraspinous fossa, between the shoulder-blades, or under them. It is usually of a dull character, uninfluenced by motion, breathing or coughing, worse during the night. The skin over the affected area is only rarely tender, but deep pressure almost invariably aggravates it; tapping this region may bring on a coughing spell. Hyperesthesia of the spine between the shoulder-blades is quite common.

[1] Ann. de Méd., 1917, **4**, 140; Progress Médicale, 1912, **28**, 234.
[2] Thèse de Paris, 1917–18, No. 63.
[3] Medizin. Klin., 1913, **9**, 1814 and 1901.

In more advanced phthisis pains in the shoulder may be actually agonizing, worse during the night, depriving the patient of his sleep, and resisting all therapeutic efforts at relief. When occurring in the incipient state they are not so acutely felt, but may extend all along the arm and forearm down to the finger tips. Minor exposures to the vicissitudes of the weather may bring about pains, and the patient then believes that he is affected with rheumatism. In fact, many cases of "rheumatism" of the shoulder turn out to be phthisis. Diaphragmatic pains are frequent. They are described by the patients as stabbing in character, or as if there was a wound in that region, and are usually due to pleural adhesions, and may be aggravated by deep breathing, coughing, and sneezing.

Hyperesthesia is very rare in phthisis unless there is complicating pleurisy. The pains are usually elicited by pressure on the regional muscles over the affected parts of the lungs. When the apex is affected, the sternocleidomastoidei and the trapezii may be painful; when the lesion is more extensive the scaleni, pectorales, and intercostals, and when there is a lesion at the base, the lumbar muscles may be painful on pressure. In pleurisy there are hyperesthesia and hyperalgesia (see p. 483). These pains are not due to cough because they are unilateral. They are accompanied by spasmodic contractions of the regional muscles, caused by reflex irritation of the supplying nerves.

These pains have been studied very carefully by Henry Head,[1] Sir James Mackenzie,[2] and more recently in this country by Lovell Langstroth.[3] Head found that these pains were either local or referred, and when due to pleurisy they coincided precisely with the situation of the pleural area involved, and were accompanied by deep tenderness, but not by superficial hyperalgesia. In cases of phthisis marked by successive acute or subacute attacks, involving previously healthy parts of the lung, referred pains were mostly found. He attributed them to the fact that the end-organs of the sensory nerves in the portion of the lung invaded remained intact, and capable of conveying impressions when irritated. These nerve endings were destroyed after the disease advanced, causing necrosis, and were no more capable of causing referred pain. Superficial tenderness is particularly liable to spread along the paths of the nerves, and Head believed it due to the cachexia and pyrexia characteristic of each acute exacerbation of the disease. Within certain limits, he was able to determine the lung area involved by the cutaneous hyperalgesia. A review of the various forms of pains in phthisis is given by F. Jessen[4] in a special monograph. Clinical experience has taught, however, that Langstroth's conclusion to the effect that this hyperalgesia is practically of no importance in diagnosis, or in localizing pulmonary lesions, is correct. But in the

[1] Brain, 1896, **19**, 153.
[2] Symptoms and Their Interpretation, London, 1909.
[3] Arch. Int. Med., 1915, **16**, 149.
[4] Lungenschwindsucht und Nervensystem, Jena, 1905.

diagnosis of pleurisy, especially of the diaphragmatic portion of the pleura, a study of the referred pains is of universal diagnostic importance (see p. 483).

It appears that the tenderness found in active phthisis is the result of the rigid contraction of the muscles—an attempt on the part of the muscles to protect the diseased viscera beneath them. It is replaced by muscular atrophy in the later stages of phthisis.

The origin of the various pains in phthisis is not always clear. It has been shown by Sir James Mackenzie that the lung is insensitive to stimulation when healthy or diseased, as is evident from the fact that when an exploring needle penetrates the lung the patient feels no pain. In fact, no form of stimulation of lung tissue seems to be capable of producing sensation, directly or reflexly. It is for this reason that necrosis of lung tissue, as it occurs in gangrene, abscess, or tuberculous cavity formation, is usually painless.

The suggestion that the pains in phthisis, as well as in pleurisy and pneumonia, are due to pleural involvement does not hold either, because the pleura is insensitive. Mackenzie states that he repeatedly explored the pleural cavity for any evidences of sensation and could employ no form of stimulation capable of producing pain. When inducing therapeutic pneumothorax I have repeatedly observed that entering the parietal pleura with the needle produced no pain, nor does scratching the visceral pleura with the point of the needle produce any sensation. Mackenzie is therefore inclined to attribute pains of the kind mentioned above to contraction of the overlying muscles. This is the reason why no hyperesthesia of the skin is met with in phthisis, but pressure pain is frequent. It is due to a visceromotor reflex, and occurs along the distribution of the sensory nerves which are stimulated by the lesion. The above-mentioned pain in the shoulder can be explained by irritation in diaphragmatic pleurisy of the phrenic nerve which conducts the stimulus to the skin of the shoulder. Both the phrenic and fourth cervical nerves leave the spinal cord at the same segment, and the former nerve conducts afferent fibers, as well as efferent (motor), and it is in all probability by the former that the stimulus is conveyed to the center of the fourth cervical nerve in the cord. Pottenger also attributes these shoulder pains to an inflammation of the nerve resulting from the reflex segmental stimulation—a true neuritis. On the other hand, investigations by Capps[1] seem to indicate that irritation of the central part of the diaphragmatic pleura gives referred pain in the neck; and irritation of other parts also gives rise to true referred pains, set up by impulses carried to the third and fourth cervical segments by the phrenic nerve, and thence to the areas of these segments. This point is discussed in detail in Chapter XXVI.

During the last few days of life the reflexes are usually abolished in the phthisical, and they are relieved from all pains; in fact, at times we find them very hopeful because they feel no more pains.

[1] Arch. Int. Med., 1911, **8**, 717.

Psychic Traits.—Psychoses met with among tuberculous patients may be considered in the main as coincidences, because so many people suffer from phthisis, and, inasmuch as this disease is no bar against mental alienation, it is but natural that some should become insane from any of the causes of this aberration. It is a fact that an enormous proportion of insane die from phthisis—Clouston[1] states that two-thirds of deaths among idiots result from tuberculosis—but this may be due to their irrational mode of life, as well as to their confinement in institutions. Delirium is also very often seen in the terminal stages of phthisis and, when not due to meningeal complication, it does not differ from the delirium seen in inanition, exhaustion, or febrile intoxication due to other causes. But in addition to these occasional psychic disturbances, which might be expected, there have been noted other psychic peculiarities in phthisical patients, and many authors have spoken of a characteristic psychology of the consumptive.

These phenomena have been observed also in infants. Combe[2] is in agreement with other authors that the tuberculotoxins act on the nervous system of infants, as of older children, and cause a decided change of character. The infant loses its gayety; it never smiles, but cries without cause. It sleeps badly, awaking often, but is difficult to arouse in the morning. This change in character is mostly observed in children with tuberculous meningitis, but is also seen in those suffering from other forms of tuberculosis.

Many tuberculous patients show a remarkable change in their mental traits and character, a disturbance in their emotional life and a striking divergence from their previous customs, habits, affections, and tastes. In some, this change precedes the evident onset of the disease, in many it appears synchronously with the symptoms of active disease; it may ameliorate with each improvement, and aggravate with each acute exacerbation.

This change in character manifests itself in various other ways: Liberal persons may become stingy and misanthropic, brave ones become cowardly, etc. Engel[3] points out that the original, innate temperament or character of the individual becomes strikingly pronounced in the chronic consumptive: The pessimist suffers from marked despondency; the optimist becomes unreasonably hopeful of the ultimate outcome, etc. These phenomena may be explained by the discordance between the subjective feelings of the patient who is not as disabled as the objective findings of the physician would lead to expect. The mental make-up of the patient depends greatly on his physical condition which, in tuberculosis, is subject to great oscillations; aggravations and improvements coming and going quite unexpectedly. The mental traits *per se* do not change, but such traits as were characteristic during youth, but, as a result of education,

[1] Allbutt's System of Medicine, **8**, 307.
[2] Le, Nourisson, 1916, **4**, 73.
[3] München. med. Wchnschr., 1902, **49**, 1383.

training, and the vicissitudes of life, have been suppressed, reappear boldly, unhindered by conventionalities.

A psychic trait of the consumptive which has been noted by most writers is selfishness. He becomes egotistical and egocentric. He is interested in the welfare of but one person—himself—to the exclusion of all who had depended on him before. He will eat costly food while his children starve; he will make unreasonable demands on his relatives and friends and show no gratitude. In sanatoriums this has been the most important problem with which the officers have to cope, and the failure of many superintendents is due to their lack of appreciation of this trait of the consumptive. As Saxe[1] states, the ascendence of selfishness plays the most important role in the molding of the mental traits of the tuberculous. In some patients these factors are so pronounced that they completely reveal the concealed elements of their characters. "They are whimsical," say Smith Ely Jelliffe and Elida Evans,[2] "have no sense of responsibility and often do not hesitate to spread infection. The nature of many adults suffering from clinical tuberculosis is that of a child, selfish, self-centered, irritable, easily angered, capricious with their food, will eat only what they like, eating irregularly, and appearing underfed. . . . Their strong infantile reactions, in that they utterly disregard others, are egotistical, dissatisfied and ungrateful." O. Amrein[3] speaks in a similar vein.

Euphoria and Euthanasia.—Optimism, despite many evidences of progressive disease which saps the body, is frequent; only a copious hemorrhage, or, more rarely, a spontaneous pneumothorax, will terrify the average tuberculous patient. Otherwise, all the symptoms amount to little or nothing. An increase in the cough is due to a "cold"; anorexia is caused by bad food, etc.

Barring the functional neuroses, there are no diseases in which suggestion—auto- and heterosuggestion—is so effective in modifying the course of the malady or in relieving symptoms. An injection of water will induce sleep, relieve pain, cough, etc., and even produce an increase in temperature exactly like that of the tuberculin reaction. In many European sanatoriums there is a routine measure before applying tuberculin for diagnostic purposes, to inject water with a view of ascertaining whether the fever is due to psychic effects, or to the tuberculin. It has been found that 20 per cent of patients react to the *injectio vacua*. Some physicians have been able to suggest the hour of the day when the reaction will appear, as well as any, or all, of the symptoms which make up the typical tuberculin reaction. The effects of this high susceptibility to suggestion are seen in phthisiotherapy; quack doctors and remedies are thriving on consumptives more than on any other class of patients, excepting perhaps the venereal, in whom the element of secrecy is of importance.

[1] New York Med. Jour., 1903, **78**, 211 and 263.
[2] Am. Rev. Tuberc., 1919, **3**, 417.
[3] Corr.-Bl. Schweitzer Aerzte, 1919, **49**, 1300.

The proverbial euphoria and euthanasia of the consumptive, which have been described in such great detail by many medical authors, and which have not escaped the attention of writers of fiction who are alert for strong dramatic effects, are other manifestations of the proclivities to autosuggestion. Experience has taught that when a patient with excessive excavations in the lungs, running high fever, and presenting other symptoms and signs of this condition, begins to believe that he has improved, that he "feels fine," has no pains, does not cough distressingly, we may look for a speedy relief of the unfortunate by that greatest of benefactors for these desperate sufferers, death. It is often astonishing to behold the sinking man making plans for the future, engage in new enterprises, plan long voyages—not for a cure, which he believes he has almost attained, but for pleasure—or, as I have seen, arranging for his marriage a few days before his death.

Very often this optimism and euphoria are excellent aids in our attempts at curing these patients. It is a well-known fact that there is hardly any hope for a despondent consumptive. On the other hand, this euphoria is occasionally harmful because it misleads the patient and he neglects the instructions of his physician.

It appears that as a result of the prolonged state of intoxication produced by the absorption of the poisons resulting from the metabolism of the tubercle bacilli, as well as of the products of decomposition of the affected lung tissue, the consumptive is in about the same mental state as those who are under the influence of mild alcoholic intoxication. The external appearance of the consumptive betrays his state of intoxication. His bright eyes with dilated pupils, which are at times contracted unilaterally, the flushing cheeks, the keen intellect which is so often met with among those who before the onset of the disease were rather dull in this respect, coupled with a flickering intelligence which brightens up suddenly for a few hours, but is soon followed by mental depression or fatigue, bear close resemblance to the average person who is under the influence of moderate doses of alcohol, or a narcotic drug.

In tuberculous patients, particularly young talented individuals, it is noted that for a few weeks or months, now and then, they display enormous intellectual capacity of the creative kind. Especially is this to be noted in those who are of the artistic temperament, or who have a talent for imaginative writing. They are in a constant state of nervous irritability, but despite the fact that it hurts their physical condition, they keep on working and produce their best work. This *spes phthisica* has been described by many authors, notably by J. B. Huber[1] and A. C. Jacobson[2] in this country. They maintain that "the quality of genius may, in some cases at least, be affected by tuberculosis," and that the intellectual powers of the genius are quickened by reason of the general psychic excitation resulting from the

[1] Consumption and Civilization, Philadelphia, 1906.
[2] Interstate Med. Jour., 1914, **21**, 341.

action of the tuberculous by-products. "They astonish everybody," says Létulle,[1] "with their mental and intellectual activity; their memory, their quick judgment, their delicate reasoning powers are of incomparable amplitude."

The long list of great writers and artists given below,[2] to which many more may be added, shows that tuberculosis is rather frequent among talented individuals, and suggests that the disease may be enhancing their productivity instead of reducing it as would be expected *a priori*.

Insomnia.—Insomnia in the early stages of phthisis may be due to restlessness owing to worry because of the diagnosis of a dangerous disease, and is often removed by emphatically reassuring the patient. Indeed, the characteristic attitude of optimism soon prevails and the patient is no more disturbed by insomnia.

In others insomnia is due to excessive cough, or nightsweats, or both. In some cases the administration of hypnotic remedies is of no avail so long as they are given in safe doses. Especially prone to insomnia are patients who suffer from paroxysmal attacks of cough, each fit waking them and keeping them awake for one-half to two hours. In these cases the administration of codein, morphin, etc., is imperative. Profuse nightsweats often act the same way: After waking bathed in perspiration, the patient finds it difficult to fall asleep again.

During the advanced stages many patients find it very hard to sleep because of the copious secretions in the pulmonary cavities which, after a short nap, overflow the bronchi and compel them to rise and expel it from the chest. Some with unilateral lesions may be able to sleep the greater part of the night in certain positions, and they adapt themselves to the conditions. But in others with cavities in both lungs, or with sinuses leading from the cavities in different directions, the prone posture immediately induces cough. Some have to sleep with the face downward if they want to avoid cough, others in the semireclining posture, etc. We also meet with cases in which dyspnea is the cause of insomnia. While during the early stages of phthisis fever may be the cause of insomnia, it is only rarely the case during the advanced stages. The average consumptive has adapted his organism to the fever and does not mind it very much. Tuberculous

[1] Arch. gén de méd., 1900, **2**, 258.

[2] It is interesting to mention some of the notable men and women who were tuberculous. Among them may be mentioned: Rousseau, Milton, Kant, Locke, Hawthorne, Keats, Shelley, Emerson, Washington Irving, Chopin, Laennec, Spinoza, Hurrell Froude, Sterne, Thoreau, Charlotte Brontë, Ruskin, Robert Pollok, Kingsley, Channing, Michael Bruce, Béranger, Thomas Hood, James Ryder Randal, Lanier, Scott, Elizabeth Barrett Browning, Bichat, Molière, Rachel, Calvin, Bastien-Lepage, Robert Louis Stevenson, Watteau, Jane Austen, Francis Beaumont, David Gray, Richard Lovelace, Georges de Guérin, Voltaire, Amiel, Paganini, von Weber, Nevins, Marie Bashkirtseff, John Addington Symonds, George Ripley, Paul Laurence Dunbar, Westcott, Blackmore, Joseph Rodman Drake, Kirke White, Stephen Crane, Adelaide Anne Procter, N. P. Willis, Henry Timrod, H. C. Bunner, John Sterling, R. Koch, Count Leo Tolstoi, Maxim Gorky, and many others.

patients with high fever are often seen sleeping quite soundly as long as the cough, nightsweats, and dyspnea do not disturb them.

In the terminal stage we often observe abnormal somnolence in phthisical patients. For days, at times for weeks, the patient lies in a semicomatose condition, careless about his person, and only now and then wakes to ask for some nourishment. If not due to excessive sedative medication, it may be an indication of meningeal complication. But I have had cases in which this abnormal somnolence has existed for several days or weeks before death, and the autopsy showed no meningeal tuberculosis. Some of these patients have periods when they are mildly delirious.

Influence of Tuberculosis on the Sexual Sphere.—The tuberculous toxemia has a profound influence on the sexual organs and their functions. In women, menstrual disturbances are not uncommon during the course of the disease, and quite often these disturbances are noted before the onset of evident symptoms of the disease. In young girls the appearance of menstruation may stay the progress of the disease, as I have seen in several cases. Probably for this reason ancient clinicians thought that amenorrhea was a cause of phthisis. Now we know it to be an effect of the disease. Amenorrhea is very frequent during the course of phthisis, and other menstrual disturbances, dysmenorrhea, menorrhagia, metrorrhagia, etc., may be observed in many cases. But I know a large number of tuberculous women in whom the menstrual function remained practically normal throughout the course of the disease.

During the menstrual days, and at times a few days before the appearance of the flow, there is often observed an aggravation in the pulmonary condition. The fever may rise, the cough increases in intensity, rales increase in number and extent, or reappear in places where they were noted before but had disappeared and new areas of lung tissue are often invaded during this period. Hemoptysis is quite frequent during this period and in rare cases it may even replace menstruation. Premenstrual fever is occasionally noted, as was already stated (see Chapter IX).

Conception is possible at any stage of the disease, and the pregnancy may, and often does, pass through almost normally, the child being of average weight but often of low vitality. Reibmeyr believes that tuberculous women are more prolific than healthy women—Nature attempts to compensate in quantity for inferior quality. Abortion and miscarriage are said to occur more often among them than in healthy women, but this has not been proved conclusively. It appears that during pregnancy the tuberculous process is, as a rule, in abeyance, and the patient may even improve. (See Chapter XXX.)

CHAPTER XIV.

INSPECTION AND PALPATION.

The Stigmata of Phthisis.—After the history and symptomatology of the patient have been carefully inquired into, the physical examination should begin with inspection of the physical make-up of the individual. In phthisis not only the chest should be carefully examined but also the head, the face, the neck, the abdomen and the extremities. The stigmata of this disease are often scattered over various parts of the body, and the experienced eye may, at times, find outside of the region of the chest certain signs which are highly suggestive of phthisis. In some borderland cases these stigmata may be of great assistance in formulating an opinion on the diagnosis and prognosis.

Complexion.—Hippocrates described the *habitus phthisicus*—the "form of the body peculiarly subject to phthisical complaints"—as characterized by a smooth, whitish skin, blue eyes, blond or reddish hair, and a phlegmatic temperament. Following this ancient clinician, many modern writers on this subject have stated that the external appearance of certain persons betrays a strong predisposition to this disease.

Hippocrates' notion that blond-haired and blue-eyed persons are more prone to phthisis has survived to this very day, and Beddoe, Landouzy, Delpeuch, Piéry, Woodruff, and many others hold the same view. Exact information, however, does not sustain this opinion that fair-complexioned people are more prone to tuberculosis. In countries with predominant blond populations, like Scandinavia, England, Northern Germany, etc., the consumptives are generally blonds; while in Italy, Spain, Greece, etc., where the dominant racial elements are brunettes, the consumptives are of the same complexion, as can be seen on visiting the sanatoriums in these countries. In China and Japan there are no blonds, yet tuberculosis is not lacking. Evidently infection, the length of time a people has been exposed to the tubercle bacilli and, above all, social and economic conditions are of greater importance in determining the rates of morbidity and mortality than race or color.

Facies.—The confirmed consumptive presents a characteristic, in fact, an unmistakable appearance, which betrays his disease not only to the experienced physician, but also to the laity, and he can often be picked out from a group of healthy people with comparative ease and certainty. The emaciated body, the pallor of the face with the hectic flush on the cheeks, the round shoulders, and the bodily decrepi-

tude, may be seen in other wasting diseases; but the facies of the consumptive, while possessing all these traits, has other characteristic stigmata. In very few other diseases is there to be seen such a pathognomonic facial expression as in the consumptive.

The facial muscles are wasted, the cheeks sunken, and the malar bones protrude; the lips are pale or livid, often contracted, as if smiling or grinning; the hectic flush, which may be unilateral; the thin neck appears longer than normal, the sternomastoids are accentuated like two tense bands on both sides; the head is bent forward between the two round shoulders, and the spine is bent. Because of the wasting, the ears appear larger; one may be redder than the other.

But the most pathognomonic parts of the cast of countenance of the consumptive are his eyes. They are deeply set in the sockets, which appear larger than normal because of the wasting of the orbicularis palpebrarum. We also meet with a widening of the palpebral aperture, and a slight protrusion of the eyeball on the affected side as a result of irritation of the sympathetic. A narrow palpebral aperture with a somewhat deeply set eyeball is a symptom of prolonged irritation of the nerve paths, and is met with in cases with adherent apical pleura, as was shown by Kuthy. To the same cause has been attributed unilateral dilatation, or more rarely, contraction of the pupil which may precede the evident onset of active disease.

The appearance of the eye as a whole is pathognomonic and can be more easily recognized than described. It has a characteristic brilliancy which has been described as transparent, lustrous, bright, dimly brilliant; it differs from the brilliancy of the eyes in other fevers in the fact that it appears gloomy, dismal, or haunted—its glance can always be felt. Some have attempted to explain these characteristics as due to the widely dilated pupils, while the pearly-white sclerotics are said to be an expression of vasomotor influence on the bulbar conjunctiva resulting from pressure on the cervical sympathetics and are to be seen mostly in cases of adherent apical pleurisy.

This facies has been recognized by the laity, and the folk-lore of Europe abounds in sayings about the facial expression of the consumptive. Writers of fiction and painters have also considered it "interesting," and have made great use of it in their productions. Many of the classical and modern painters have depicted this cast of countenance, showing the false euphoria of the smiling, tranquilly bright, yet melancholy eyes of the consumptive, which are perhaps best seen in Leonardo da Vinci's *La Gioconda*—a picture of a phthisical face superior to any description that can be given of it.

I have seen these facies in some patients with latent, or quiescent, tuberculosis in whom physical exploration of the chest showed but indefinite signs of an active lesion. It appears to be especially marked in persons of phthisical stock and in young individuals with glandular tuberculosis, those who were infected during childhood, but have more or less recovered.

The Skin.—Other stigmata of phthisis, which may be noted in the early stages of the disease, should be mentioned. On the forehead and upper parts of the cheeks we may see *chloasma phthisicorum*, and in those who sweat profusely, *pityriasis versicolor* and *tabescentium* on the anterior and posterior aspects of the chest. In those who suffer from dyspnea, we may find clubbed fingers, or deformities of the hands, wrists, spine and tarsus, which are the results of pulmonary osteo-arthropathy. On the neck, spasm or atrophy of the muscles, which will soon be described, may give a clue that a careful examination of the chest is indicated.

Enlarged Glands.—Visibly enlarged glands are quite rare in adults, though I have seen cases in which they went on to suppuration. But palpable glands on the neck are very frequent—in at least 50 per cent of my cases. In children, enlarged glands are very frequent, but they are not always an indication of tuberculosis. If enlarged cervical glands were pathognomonic of tuberculosis in children, we should find very few who live in poverty free from this disease (see Chapter XXIV). Of greater importance from the diagnostic standpoint is enlargement of the supraclavicular glands, especially when found unilaterally. It speaks for tuberculosis of the costal pleura.

We also very often find enlargement of the thyroid gland in tuberculous subjects, at times in the incipient stage, and mild grades of exophthalmos are not uncommon. The reciprocal relation between hyperthyroidism and tuberculosis is discussed in Chapter XXX.

Enlarged Veins on the Chest.—Enlarged veins are often seen on the chest, especially in the infraclavicular region over the first and second interspaces, and posteriorly opposite the first thoracic spine, and below along the line of insertion of the diaphragm. The upper enlarged veins are caused by the interference with the emptying of the internal mammary and intercostal veins, because of pressure on the vena azygos by swollen thoracic glands, and also by the increased expiratory efforts while coughing. They are occasionally seen in healthy persons, especially in nursing women, and they may be unilateral in patients suffering from chronic bronchitis and pulmonary emphysema, as well as with endothoracic tumors. According to Lombardi,[1] the varicosities in the neighborhood of the seventh cervical and first thoracic vertebræ may be seen in 80 to 90 per cent of cases of phthisis, but I see them very frequently in persons without any active pulmonary disease.

It will also be noted in some cases that the nipple is located lower or more externally, while in women the mammary gland may be smaller, and the nipple may be less pigmented, than on the opposite unaffected side.

The Normal Chest.—Before looking for pathological changes in the form and structure of the chest we must have a clear idea as to what constitutes a normal thorax, and it must be stated at the outset that

[1] Gior. internaz. di Sci. med., 1913, **35**, 751.

a normal, well-formed chest is an ideal which is not encountered more often than physical perfection in general.

The chest in a normal person is symmetrical; the two sides are practically alike. Both clavicles run horizontally, are not very prominent at their external third; only their contour is visible. The supra- and infraclavicular fossæ are only slightly deepened, and both to the same degree. When the arms hang by the sides the scapulæ lie flat on the back of the chest extending from the second to the seventh ribs; in rare instances they are lower, from the third to the eighth ribs. The supra- and infraspinous fossæ are filled out with muscles. The spinal column runs in a straight vertical line, showing a slight convexity backward at the upper half of its dorsal portion. The ribs run downward and forward, are hardly visible in the upper third of the chest because they are covered with muscles and subcutaneous fat, while in the lower third they are clearly prominent, and the intercostal spaces are perceptibly indented. Anteriorly the costal arches form almost a right angle, between 60 and 80 degrees; the sternum is straight in profile, about 16 to 20 cm. in length, and Ludovig's angle, at the junction of the manubrium and gladiolus, is hardly visible. In some persons enjoying perfect health, Harrison's groove, denoting the insertion of the diaphragm, runs horizontally, commencing at the lower end of the sternum and passing downward to the axillary line. The two shoulders are of the same height; so are the nipples. The antero-posterior diameter is somewhat shorter than the transverse. All the diameters of the chest should increase from above downward, giving it the appearance of a pyramid with its base below. But this pyramidal form is not very evident in the average healthy person because of the muscles, female breasts, and the clavicles over its apex. During the first year of life the transverse diameter is almost as large as the antero-posterior; a transverse section appears almost round (Fig. 15). With the growth of the child, this infantile form of chest changes, the transverse diameter increasing in length, so that in a child of ten, a section would appear elliptical, because of the various projections of muscles, etc.

The ideal chest, seen perhaps only in the works of great sculptors, is, of course, rarely seen in practice because of slight hypertrophy of the muscles on the right side in those who work hard, slight shoulder droop in those who have minor forms of scoliosis, etc., all of which are consistent with perfect health of the thoracic viscera.

While the ideal chest is only rarely seen in healthy persons, it is never seen in tuberculous patients. In the latter, going hand-in-hand with the progress of the disease, the form and shape of the thorax change as a result of certain changes in the respiratory muscles, and in many cases we find on inspection and palpation conditions which are characteristic of the phthisical chest.

The So-called Phthisical Chest.—Beginning with Hippocrates, Galen, and Aretaeus, physicians and the laity have for centuries associated certain forms of abnormal appearance of the chest with tuberculosis, or

with a predisposition to this disease. It has thus come about that nearly all deformities of the chest excite suspicion of active or latent tuberculosis. The fact is that there is no form of the chest which is pathognomonic of pulmonary tuberculosis, though there are certain changes in its appearance which point to this disease.

The *flat chest*, the *paralytic chest*, and the *pterygoid* or *alar chest* are very frequently seen in youthful tuberculous subjects, though they are also often seen in individuals who pass through life without developing tuberculous disease of the intrathoracic viscera. In the flat, or paralytic, chest the ribs run steeply downward toward the sides, and the result is that the anterior portions of the ribs and cartilages appear flat

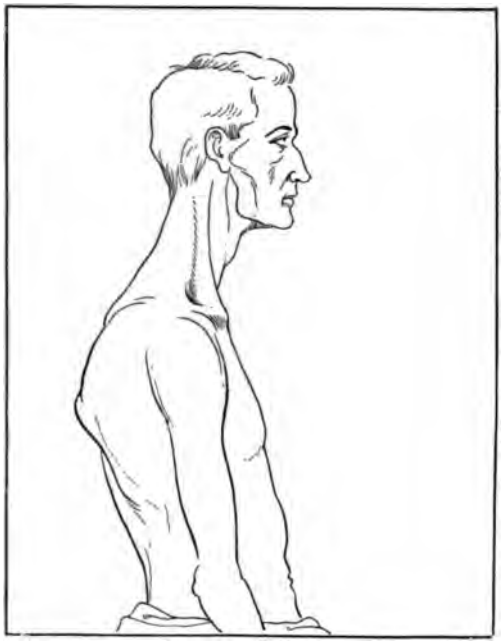


FIG. 54.—The "phthisical" or flat chest. Habitus phthisicus.

instead of convex; in other words, the chest appears as if it were in a state of extreme expiration. It is very long, narrow, and flat, the intercostal spaces appear narrower but excessively deepened, especially over the lower half of the chest. The antero-posterior as well as the transverse diameters are shorter than normal, the scapulæ move to the sides, and appear winged as a result of the reduction in the space available for them on the back of the chest, and also because of atrophy and loss of tonicity of the muscles, especially the serrati. The clavicles are very prominent, and the fossæ above and below these bones are strikingly deepened. As was already stated, Freund insists that in this sort of thorax the first costal cartilage is shorter than normal, only 3 cm.

long in men and 2.2 cm. in women as against 3.8 and 3.1 cm. respectively in normal persons. This shortening, together with ossification of the first cartilage, produces a narrowing of the upper thoracic aperture, which is said to be a strong predisposing factor to tuberculous disease of the lung apex. When other costal cartilages are also ossified, as is often the case in phthisis, the elasticity of the bony framework of the thorax is impaired and expiration is interfered with. The shoulders

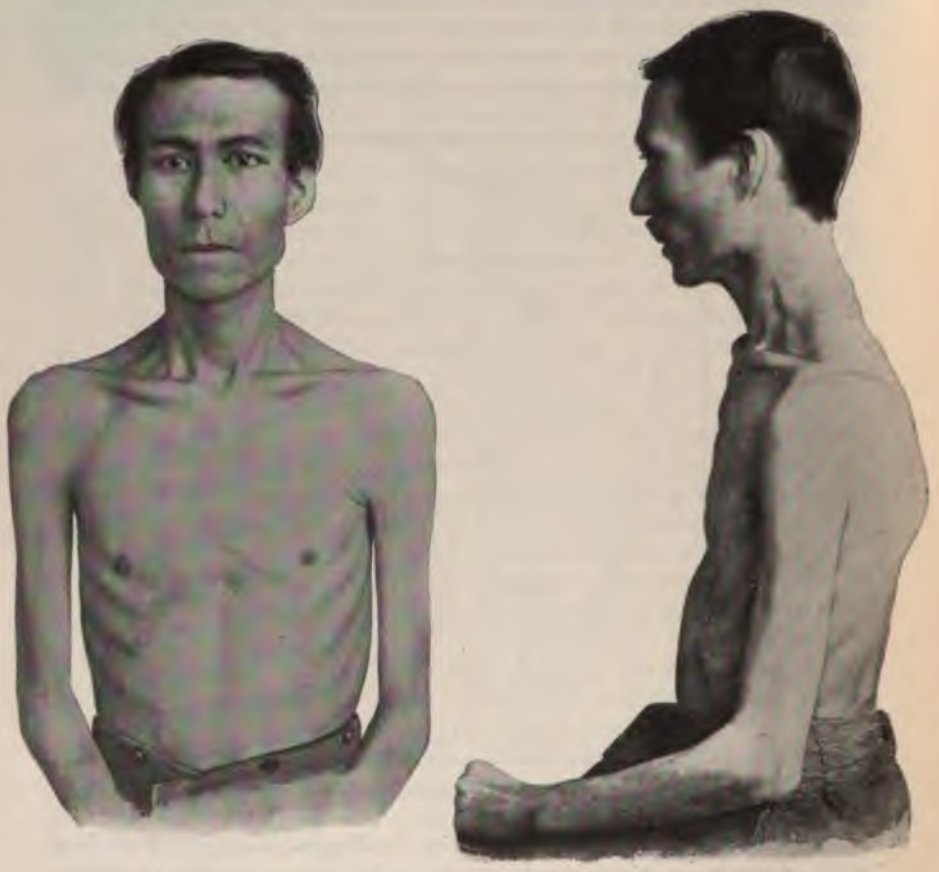


FIG. 55.—The "phthisical chest." Full-blooded Indian. (Musser.)

are usually sloping on both sides, the attachment of the ribs to the sternum is at an acute angle, and they run obliquely downward to the sides. The inferior costal margins reach down very low, almost to the crest of the ilium in some cases, the subcostal angle is very acute, reaching 25 degrees in some instances, and the angle of Louis appears as a strongly marked ridge. The spinal column usually shows a strong kyphotic bend of the dorsal portion without compensating lordosis of

the lumbar part. Because of the lack of subcutaneous fat, and the atrophy of the muscles of respiration, some have called this form of chest the *asthenic*.

There are to be distinguished two forms of this chest, the congenital and the acquired. The congenital may be hereditary, when occurring in newborn infants from tuberculous parents, but this is rare. It is mostly seen in infants of poor constitution, in the rachitic and undernourished. In adults it is seen mainly in individuals whose muscular system is poorly developed, thus presenting evidences of the asthenic constitution. The intercostal muscles being weak, inspiratory efforts are feeble and do not expand the chest properly. It is thus said to be an expiratory chest. Practically, it will be noted that in most cases only the upper part of the chest does not breathe properly.

It is not wise to condemn a person with this type of chest to tuberculosis, or even to see in it a strong tendency to this disease when no constitutional symptoms are found. Many, or most, persons with the asthenic constitution never develop tuberculosis. There seems to be available evidence to the effect that this asthenic chest is the result of ancient tuberculous infection, especially during childhood, affecting the intrathoracic glands, and that the lesions have healed. Elsewhere in this book we show that these tuberculous infections have a certain immunizing effect against the development of active phthisis in the adult.

With the acquired form of the flat chest things are different. Persons with this type of chest have either active, or quiescent tuberculous lesions in the lungs. In most cases it will be noted that the symmetry of the chest is more or less disturbed; the muscles of respiration, the trapezii, the pectorales, the rhomboidei, the scaleni, etc., are unduly contracted when the tuberculous lesions are early, or atrophied on one side of the chest with advanced or healed lesions. The diaphragm, the only muscle to act during respiration, is often immobile on one side owing to pleural adhesions and destruction of the pulmonary parenchyma. One scapula is more winged than the other, or nearer the middle line. One shoulder is lower than its mate, and there are seen retractions of the chest wall on the side which is most affected. These changes are discussed further on in this chapter.

Technic of Inspection and Palpation of the Chest.—In addition to the light, warm room and stripping the patient to the waist, which are self-evident requirements, the patient is to be seated on a round stool, directly facing the window or the source of artificial light. He is permitted to assume his natural posture without urging him to sit straight up, hold his head in the middle line, etc., so that we may note any faulty position of the head, neck, spine and chest. Careful attention is to be paid to the position of the head, the shoulders, the clavicles, the ribs and the scapulæ during rest, during moderate, and forced breathing.

Above all, *we are looking for evidences of asymmetry in structure,*

form, and mobility, when the two sides of the chest are compared. Motion can be ascertained by inspection, carefully noting from a distance the tips of the acromion processes, as well as the elevation of the ribs during inspiration, the position of the scapulæ during both phases of the respiratory act, and also the lateral expansion of the lower parts of the thorax. Flattening, excavations, and undue prominence of the

FIG. 56.—Emphysema with enlargement of the chest; the antero-posterior diameter is much increased. (Musser.)

respiratory muscles are to be especially looked for. The supraspinous and supraclavicular fossæ are compared, and no deviation from the normal should be overlooked. Spinal deformity, if present, must be given attention, for it may be the result of an intrathoracic lesion; because it may have an immense influence on the interpretation of the results obtained by percussion and auscultation, and also on the roentgenogram.

The motion of the anterior aspect of the thorax is well studied while standing behind the patient and looking over his head, watching the ribs and clavicles as they rise and descend during inspiration and expiration, and noting any retardation or limitation of motion on one side as compared with the other. It is, however, best to ascertain this by palpation, placing the hands on each side of the patient's neck, the thumbs meeting behind at the spine and fingers reaching down over the clavicles (Fig. 57), and for the lower parts by placing the hands over the lateral aspects of the chest. In this manner slight differences can be detected more easily than by inspection. Special attention is to be paid to lagging—one side of the chest is delayed in

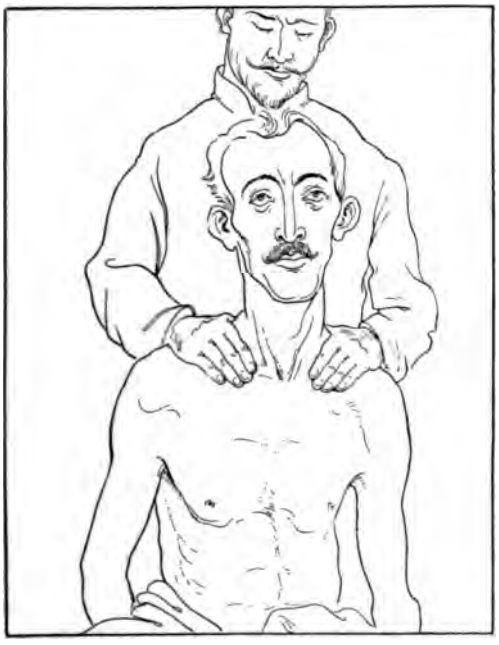


FIG. 57.—Testing mobility of the chest.

movement and, in more advanced cases, expansion is limited. At times we meet with both lagging and limitation of motion in various parts of the chest and we may conclude that the former is an indication of a recent lesion, while the latter is caused by an old, probably pleuritic lesion.

Spasm and degeneration of muscles of the neck and chest are best ascertained by Pottenger's method of "light touch palpation." Pressing the tips of the fingers over the muscles under consideration and moving the hand sidewise, carefully noting the degree of resistance, will show this condition. While doing this the fingers should not be allowed to slip on the skin, because it is the condition of the muscles,

and not of the skin, that we wish to ascertain. Over acute lesions it is found that the muscles give to the palpating fingers a distinct feeling of increased resistance, that they are firmer and fuller than normal, while over advanced lesions there is a flabby, doughy feeling, and the bundles can be easily separated owing to atrophy and degeneration.

Significance of Lagging.—In the very incipiency of a pulmonary lesion we often note that the affected side of the chest begins to expand, and the shoulder to move upward, later than the opposite healthy side of the chest, and finally does not attain the same amount of expansion. In far-advanced cases and in those with pleural adhesions and effusions, pneumothorax and intrathoracic tumors, there may even be absolute immobility of the affected side. It is best ascertained by letting the patient first breathe normally and then asking him to take a few deep inspirations.

Lagging of the upper part of one side of the chest is an indication of a lesion in that apex, provided an acute or chronic non-tuberculous inflammatory process of the lung and pleura is excluded. When the motions of both sides are equal, but there are sure signs indicative of tuberculosis, we may conclude that there is a bilateral lesion. With an old quiescent lesion in one side and a new and active lesion in the other, the lagging is more pronounced in the newly affected side. I often find difficulties in clearing up by inspection and palpation old bilateral lesions in which both sides show limited motion. In these, percussion and auscultation give more reliable information. But in incipient unilateral cases inspection is of immense value.

Thoracic Asymmetry.—Looking at the phthisical chest anteriorly, in cases in which the disease has already made some inroads, we find some undue prominence, even arching of the clavicle and more or less deep excavation in the supra- and infraclavicular fossæ, more marked, or exclusively, on the affected side. The angle of Louis at the junction of the manubrium and the gladiolus is more pronounced than in the average healthy chest. Posteriorly, we find kyphosis in many cases, the scapulæ are prominent, winged, and even dislocated, nearer the spine on the affected side. The intercostal spaces are narrow, but deeply indented and, in extreme cases, the free margins of the costal cartilages nearly meet in the middle line. In addition to these changes we meet with distortions of various parts of the chest, especially the upper half—flattening and retractions of various degrees anteriorly and posteriorly. Depression of the acromial end of the clavicle on the affected side may be already noted in the very early stages of the disease. Kuthy[1] found it in 82 per cent of his incipient cases.

Inspiratory Retractions.—In patients with far advanced lung lesions localized inspiratory retractions may be seen on the chest wall. In some instances in which large cavities exist in the upper lobe we may note during each inspiration a cup-shaped depression of the intercostal

[1] Sixth Internat. Congr. Tuberc., 1908, **1**, 1215.

space covering the cavity, usually the second, third or fourth interspace. In extensive left-sided lesions pulsation of the displaced heart may be visible owing to retraction of the lung and pleural adhesions. When there is great difficulty in the entry of air into the alveoli, as is the case in acute pulmonary miliary tuberculosis, and pulmonary edema, the lower margins of the chest sink in quite deeply during each effort at inspiration. This is especially to be observed in children and in young adults in whom the chest wall is elastic and yielding. In emphysematous individuals, and in patients with tuberculous lesions in the upper lobes, but in whom the lower lobes are vicariously emphysematous, there may be seen inspiratory retractions of the lower margins of the chest, because the diaphragm does not move properly and the lungs do not fill fully with each inspiration.

FIG. 58.—Muscles of the neck which are either spasmodically contracted or atrophied in pulmonary tuberculosis.

Localized inspiratory retractions are excellent signs of airless tissue beneath the retracted area. Whether the airless tissue is tuberculous, pneumonic, or atelectatic in origin, or due to pleural adhesions, should be made out by a study of the concomitant symptoms and signs. Retractions are not to be confused with immobility of a part of the chest wall which may be due to pleural adhesions, pain in the chest, disease of the muscles, pleural effusions, intrathoracic tumors, etc.

Spasm and Degeneration of the Thoracic Muscles.—Any, or most, of these changes in the contour of the chest may be noted in cases of non-tuberculous affections of the thoracic viscera, and also in patients who had tuberculous lesions which have healed, the patient being in excellent health. Pottenger, in his epoch-making studies of the tuberculous chest, has given us certain clues as to the means of differentiating these conditions. It appears that intrathoracic conditions

have a great influence on the muscles of respiration, a fact which has been known for a long time, but was rationally interpreted and made available for diagnosis by Pottenger.

Whenever the lung or pleura is acutely inflamed, the thoracic muscles over the seat of the lesion are in a state of spasmodic contraction, like the abdominal muscles in a case of appendicitis. Depending on the acuteness of the inflammatory process in the pulmonary parenchyma or pleura, the muscles of the neck and chest show this contraction in various degrees. As in other visceral inflammations, this is an involuntary reflex protective spasm.

Inspection and palpation reveal this condition very clearly in the vast majority of cases. Muscles in spasm are larger and firmer in appearance as well as to touch, giving a distinct feeling of increased tension. Often the more tendinous parts of muscles feel like distinct cords, while the more fleshy parts are larger and firmer to the touch than normal muscles on the opposite unaffected side.

After the inflammatory process in the lung and pleura has lasted for some time, and passes into a chronic stage, the muscles degenerate; they waste and become flabby. To the palpating finger they feel doughy, their normal tone and elasticity are gone, and their bundles are easily separated. It is important to note that, coincident with this change in the muscles, there is always seen atrophy of the skin and disappearance of the subcutaneous tissue. Some of these changes are evident to the sight as well as to the touch.

Pottenger looks upon these muscle changes as due to reflex stimulation of the motor nerves, the result of continuous irritation caused by the impulse from the inflamed lung and pleura. When this irritation is kept up very long degeneration and wasting follow, though the latter may be due partly to trophic disturbances. But if it is true that we can make out by superficial palpation of the dead body internal solid structures, as some have maintained, it would indicate that the theory of reflex irritation is inadequate.

Muscular Changes in Incipient Cases.—In incipient cases we often find that the sternocleidomastoid, the scaleni, and pectoralis anteriorly, and trapezius, levator anguli scapuli, etc., posteriorly, are in a state of spasm: They stand out more prominently, are larger and firmer to the touch than the same muscles on the opposite, unaffected side. I have often seen that as a result of this spasm the supraspinous fossa was fuller at first sight. When occupational influences can be excluded, it is a good sign of active incipient phthisis. When combined with lagging of the same region, or, at the base of the same side, it is undoubtedly a sign of a lesion of the lung, provided non-tuberculous disease can be excluded. To distinguish these changes in the muscles from those resulting from occupational influences, it is to be borne in mind that the sternocleidomastoid muscles rarely, if ever, hypertrophy or waste from overuse, or disuse, nor does the subcutaneous tissue show any changes.

Muscular Changes in Advanced Disease.—With the advance of the disease, the affected muscles, as a result of prolonged spasm, begin to atrophy and degenerate. The result is that on inspection and palpation even better criteria of the intrathoracic condition may be elicited. The degeneration of the skin and subcutaneous tissue over the site of the lesion is seen at once; the skin can be lifted up with the fingers more easily, and it is felt that it lacks the normal elasticity. The sternocleidomastoid, scaleni, pectoralis, trapezius, levator anguli scapulæ and rhomboidei all look smaller than their mates on the unaffected side. They are flabby and doughy to the touch.

In cases with old circumscribed lesions limited to the upper part of the apex we may find the upper half of the pectoralis degenerated and flabby, while the lower half is normal. As a result of atrophy of the trapezius we find flattening of the supraspinous fossa; in extreme cases it appears cupped. In old cases extension of the disease may often be ascertained by inspection and palpation. The old lesion on one side shows wasting of the skin and muscles, while on the opposite side, where tubercles have just formed a new incipient lesion, the muscles are in spasm—contracted and prominent. Lagging is more pronounced on the newly affected side; it indicates an active lesion which hinders motion of the contracted muscles, especially the diaphragm. "When palpation, percussion and auscultation show evidences of a lesion and there are changes in the mobility of the suspected side and no spasm of the muscles over the apex but, on the contrary, the tone of the overlying muscles has decreased, and there are evidences of atrophy of the subcutaneous tissue combined with clinical symptoms of tuberculosis, we are justified in concluding that we deal with an old, inactive, or healed process." (Pottenger.)

In many cases we may find the regional muscles more or less atrophied from disuse, especially when compared with the opposite side, where they are enlarged, firm, and prominent because of excessive occupational hypertrophy. This is best differentiated by bearing in mind that in muscular atrophy due to disuse, the subcutaneous tissue is normal, while when due to a pulmonary lesion it is atrophied.

Effects of Muscular Atrophy on the Thorax.—Lagging, which was formerly attributed to lack of expansion of the affected lung or pleural adhesions, is better explained by the tonic contraction of the scaleni and sternocleidomastoid on the affected side, which raise and fix the sternum, and immobilize to a certain extent the first and second ribs, thus limiting the respiratory motion of the affected side. Round shoulders, which were formerly attributed to weakness of the posterior muscles which hold the spine erect, are more rationally explained by Pottenger as due in a great measure to shortening of the anterior muscles through spasm and degeneration, together with lessened mobility of the thorax. Flattening of the chest, especially over pulmonary cavities, which was formerly attributed to atmospheric pressure forcing the bony thorax inward, in order to occupy space

previously occupied by lung tissue, is explained by Pottenger as due to inflammatory disease within the thoracic cavity, and reflex interference with the normal motion of the diaphragm, which is known to be part and parcel of phthisis from roentgenographic studies.

Bearing in mind that the vast majority of persons are infected with tuberculosis during childhood, but that the pulmonary lesions heal, or remain latent, it is understood that the lesions produce muscular changes in the manner described above during the time of their activity. Thus, we have an explanation for the origin of the phthisical or paralytic thorax. It is a result of an earlier infection which has healed or remained latent and quiescent and is not a predisposing cause of phthisis. A careful study of children of tuberculous parentage has shown that, as a rule, they are born with normal chests, and the characteristic deformity only occurs later in life after infection with tubercle.

Vocal Fremitus.—Palpation for the *vocal fremitus* is of but little diagnostic value in any stage of phthisis, excepting in cases where pleural effusions are suspected. But it is often absent in thickened pleura and intrathoracic neoplasms and thus is not of great assistance in our attempts at differentiating the latter from an effusion. Of course, it is increased over consolidations of lung tissue, and when due to tuberculosis, consolidation may thus be elicited by palpation for the vocal fremitus. But the consolidation must be extensive, involving the greater part of a lung, to be easily discovered by palpation. In early lesions the writer's experience has shown that percussion and auscultation elicit definite signs, but the vocal fremitus remains normal in most instances.

Myoidema.—In some cases of tuberculous disease of the lung myoidema may be observed—a nodular swelling of a muscle produced by tapping it with the finger, or filliping it, which causes a local contraction lasting several seconds and disappearing. It should not be confused with another kind of muscular contraction induced by percussion, the *fascicular*, consisting in a deep furrow along the entire length of the muscle; in myoidema merely a nodular swelling is produced; hence it has also been called *nodular contraction*. It was first described by the great Irish clinician William Stokes in the *Clinical Report of Cases in the Medical Wards of the Meath Hospital during the Session of* 1828 *and* 1829.[1] "Some time ago, on percussing a patient (immediate percussion was then practised) who had labored under a pectoral affection, with several symptoms indicative of tubercular development, we were surprised to observe that after each stroke of the ends of the fingers a number of little tumors appeared, answering exactly to the number and situation of the points of the fingers where they had struck the integuments of the chest. These having continued

[1] Dublin Hospital Reports, vol **5**, quoted from Stokes's Treatise on the Diagnosis and Treatment of Diseases of the Chest, New Sydenham Society, p. 429.

visible for a few moments, subsided; but could be again made to appear on repeating the percussion."

This sign was practically forgotten until recent years, when several authors began to publish papers on a new sign of tuberculosis consisting in hyperexcitability of the muscles. Verrienti[1] says that this muscular hyperexcitability is a phenomenon which is present in all stages of phthisis, is most marked on the side with the most active lesion, not necessarily where the changes are most extensive. All the muscles of the body may thus react to percussion, but those of the thorax, especially the supraspinatus, pectoralis major, and the trapezius, show it in a most pronounced manner. It is attributed to supersaturation of the body with the toxins of tuberculosis, especially the muscles, the smooth as well as the striated. Flushing of the cheeks, turgescence of the veins, diarrhea without tuberculous ulcerations of the intestines, irritability of the heart muscle, etc., which are observed during the course of phthisis, are all due to this muscular hyperexcitability. Halbron, Pradal, Sainton,[2] F. Levy, Theodorescu,[3] and many others, are inclined to attribute great diagnostic and prognostic significance to this myotonic reaction. They point out that it is absent in chronic bronchitis and in pulmonary emphysema, and when these conditions are complicated by tuberculosis, rendering it difficult to localize the lesion, the side in which the tuberculous process is located may be discovered by detecting myoidema.

But Stokes already, in 1828, pointed out that myoidema is not altogether pathognomonic of phthisis. He said: "There is nothing in this muscular irritability peculiar to phthisis, but that it is commonly connected with irritation of the lung, or pleura, there can be no doubt; and in this way, like other signs of irritation, it becomes available in the diagnosis of phthisis. It is always more evident in the earlier periods; thus in incipient phthisis it occurs over the primary seat of irritation, while in the confirmed and chronic cases we may often find it absent over the diseased lung, and strikingly marked on the side last and least engaged." More recently S. West[4] investigated this sign and found it present in tuberculous patients, and also in many others, healthy, as well as sick with various diseases involving emaciation, or absence of subcutaneous fat, which is one important factor in its production. In some people it is probably physiological. Many recent writers have found it in numerous morbid conditions of toxemia and nervous fatigue, such as plumbism, alcoholism, cancer, various infections, notably typhoid fever, lethargic encephalitis, acute articular rheumatism, nephritis and uremia, Addison's disease, etc.

Myoidema can therefore not be considered a sign pathognomonic of tuberculous disease of the lung or pleura.

[1] Riforma Med., 1919, **35**, 571.
[2] Bull. et mém. Soc. méd. d. hôp. de Paris, 1919, **43**, 980.
[3] Ibid., p. 973.
[4] Diseases of the Organs of Respiration, London, 1909, p. 510.

CHAPTER XV.

PERCUSSION OF THE CHEST IN PHTHISIS.

WHILE the value of percussion in the diagnosis of conditions in the advanced stages of phthisis, and its complications, is not questioned, it has been very seriously debated whether it can give dependable information in the early, or incipient, stage. Many authorities, notably of the French school, like Grancher, Bezançon, Barbier, Piéry; and also S. West, Bonney, Lawrason Brown, Henry Sewall and others maintain that small tuberculous foci in the lung in incipient phthisis can be recognized solely through recourse to auscultation, and that when dulness is elicited on percussion, we may be confident that we are dealing with extensive infiltration—a more or less advanced stage of the disease. On the other hand, Aufrecht, Krönig, Goldscheider, William Ewart, Lees, Riviere, and many others, maintain that if we are to detect incipient lesions in phthisis, we must resort to percussion, and it is only when the process has advanced that definite auscultatory signs are elicited.

Aims of Percussion.—It seems that these differences of opinion are mainly due to a misapprehension as to the aims of percussion. Those who expect to make a diagnosis relying solely on percussion findings will be sadly disappointed, just as they will fail in attempting to draw final conclusions from any other single symptom or sign. *Percussion only gives information about the density, or the air content, of the lung at the point examined.* Whether an airless area thus detected is due to a tuberculous infiltration, or to one of the numerous other factors that may consolidate large or small areas of lung tissue, must be determined by a study of all the concomitant symptoms and signs. On the other hand, given constitutional symptoms of phthisis such as cough, fever, anorexia, etc., signs of a limited infiltration, or of a circumscribed area of airless lung tissue, elicited on percussion, may enable us to localize the process and complete the diagnosis in the absence of auscultatory signs.

We must bear in mind that phthisis does not begin as a catarrh of the small bronchi, as some believe, but as an infiltration, transforming the normal porous, air-containing, and resonant lung into solid non-resonant tissue. At this stage the alveoli are filled with exudate, or the interstitial tissues contract and compress the alveoli, finally obliterating them altogether. Inasmuch as altered breath sounds and rales can only be found in the pulmonary apices when edema and secretions interfere with the entry or exit of the air current while passing through the air vesicles and bronchioles, it is clear that auscul-

tation may not give any information at a very early stage. So long as the infiltration remains beneath the mucous membrane of the bronchi, the entrance of air into the alveoli of the affected area is not interfered with very much, while in the rest of the lung it is freely circulating. Auscultation may not reveal such a lesion which is surrounded by healthy lung tissue working vicariously and sucking in more air.

It is only when the caseous material of the infiltrate softens and breaks through the wall of a bronchus, thus permitting the entrance

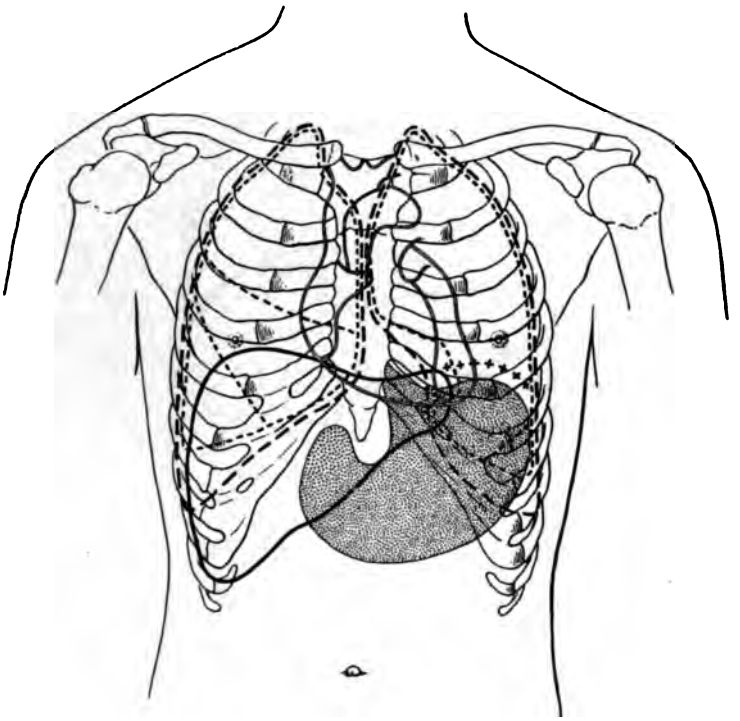


FIG. 59.—Outlines of viscera. The margins of the lobes of the lungs are shown (interrupted line------); solid black line, heart, liver, and spleen; stomach shaded. (After His-Spalteholtz, Luschka, and Musser.)

of air into the diseased focus proper, that rales can be heard on auscultation. At that time tubercle bacilli make their appearance in the sputum. When we have rales we may be sure that we are dealing with a more or less advanced stage of the disease—caseation and softening have already taken place.

When the tuberculous process was not located originally in the bronchioles, but in the peribronchial tissues, it is again evident that the air circulating in the bronchial tree cannot reach the tubercle at all, and the auscultatory signs will necessarily be negative. At most, feeble,

or the absence of breath sounds over a limited area may be the first sign elicited.

Technic of Percussion.—Percussion has been neglected by many because it has not given them the information they sought; at times it even misinformed them. The reason is almost invariably faulty technic. Before giving details as to percussion findings in early phthisis, we must speak about the proper technic to be followed in apical percussion.

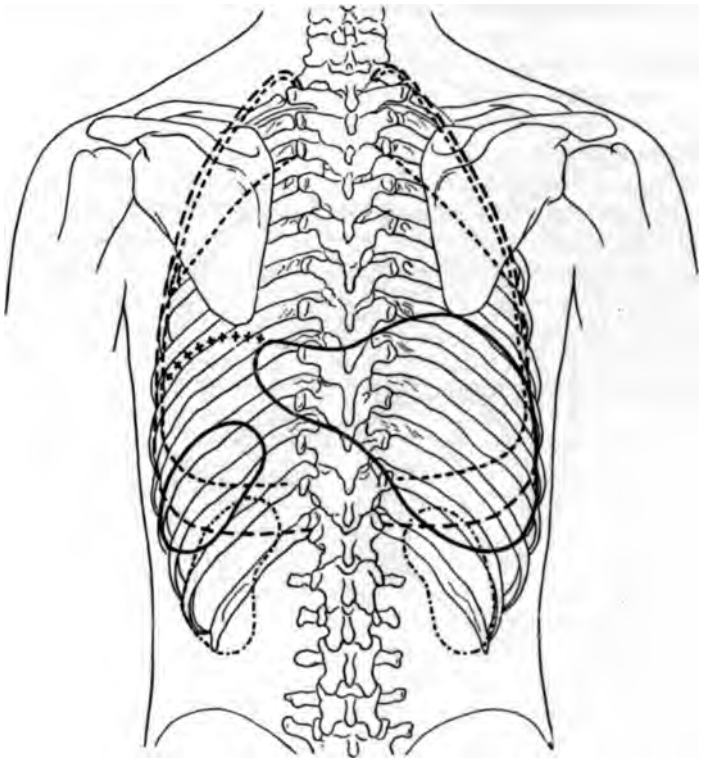


FIG. 60.—Outlines of viscera. The margins of the lobes of the lungs are shown (interrupted line ------); solid black line, heart, liver, and spleen. (After His-Spalteholtz, Luschka, and Musser.)

The first and most important point in percussion is a light stroke with the finger. Heavy blows with two or three fingers are worse than useless. Because of the elasticity of the thoracic walls, a great part of the percussion stroke is always dissipated along the muscular and bony parietes, and when we strike a heavy blow most of the force is conducted laterally by the ribs and intercostal muscles, which are set into strong vibration, acting as large pleximeters, and resonance from the entire lung beneath is elicited. Small areas of airless tissue are thus overlooked. With a light stroke the force is not conducted along the

parietes, but penetrates sagitally into the lung, affording information about its condition immediately beneath the point examined.

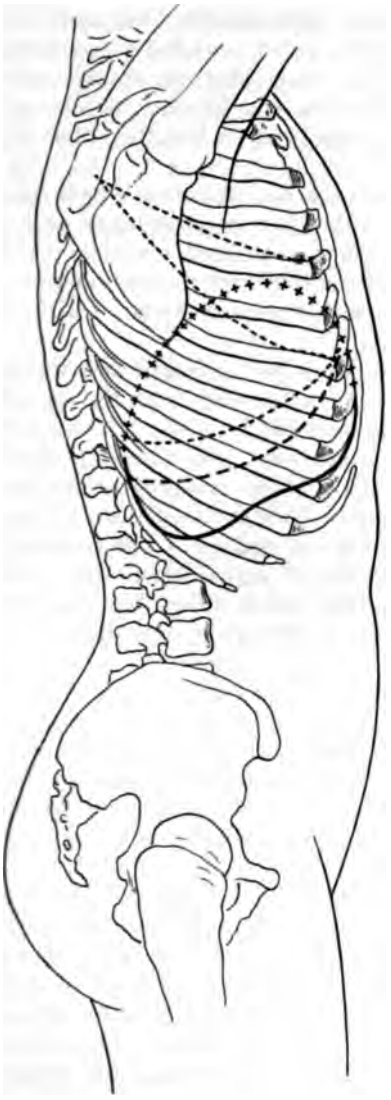


FIG. 61

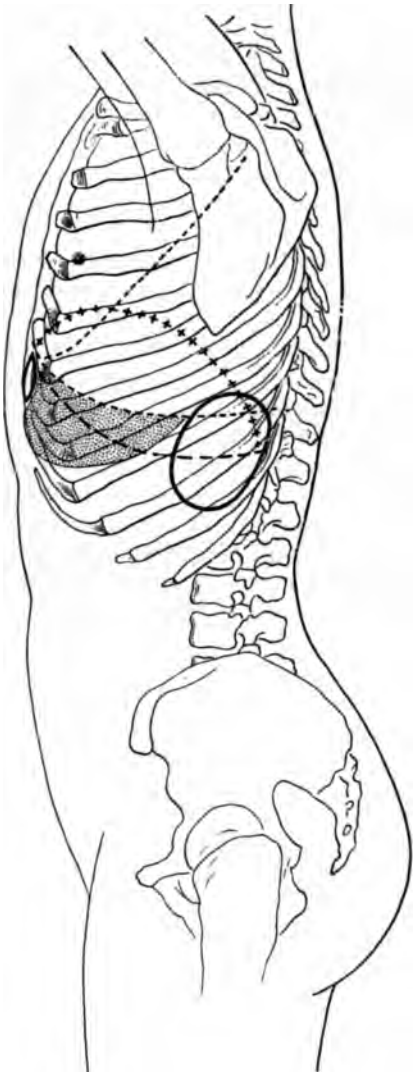


FIG. 62

FIGS. 61 and 62.—Margins of the lungs and of individual lobes, dotted line (- - - - - -); limits of pleural sacs, interrupted line (- - - - - -); liver and spleen, solid black line; diaphragm, starred line (******); stomach (portion not covered by lung) shaded. (After Luschka and Musser.)

With light percussion in which the stroke is gentle and soft, hardly audible at any distance, we can always localize areas of superficial

dulness. Deep-seated, airless areas cannot be detected by heavy percussion, as is evident from the fact that we cannot map out the heart from behind, and in obese and edematous persons it is quite difficult, often impossible, to define the boundary between the liver and the lung. Strong blows do not reach much deeper into the pulmonary tissue proper than light strokes. To be sure, they set up stronger vibrations, but mainly in a lateral direction, and for this reason the penetrating power of the heavy blow may be even less than that of the light stroke.

Gentle percussion often brings out small areas of dulness which disappear with an increase in the force of the blow because larger areas have been set into vibration. This point is utilized for diagnostic purposes: If, on increasing the force of the blow, the dulness remains, we may be sure that we are dealing with extensive areas of airless tissue.

The Plexímeter Finger.—Light percussion is best accomplished when the movement of the percussing finger is exerted only from the metacarpophalangeal joint. The note elicited should be only a faint sound which can be heard when listening attentively. Of course, perfect silence must be maintained in the room. When reaching an airless area, the contrast between the resonance evoked in the air-containing space and the deadness over the dull area is striking. The contrast between something and nothing is easier of appreciation than the difference between one thing and another which differs but slightly from it. Over resonant areas we evoke a note, while over dull areas no note is brought out at all.

Strong pressure of the plexímeter finger on the chest wall dissipates the advantages of light percussion by bringing the intercostal muscles into tension, making them large plexímeters, which elicits resonance of the neighboring air-containing lung, and small areas of dulness can thus not be delineated. Very light contact of the plexímeter finger with the chest wall is therefore important; in delicate percussion, the mere weight of the finger is sufficient.

Bearing in mind that, as a rule, tuberculous lesions spread from above downward, and that the line between the healthy and infiltrated tissue usually runs horizontally, we must percuss from above downward, or the reverse, in vertical zones. *The plexímeter finger should be placed parallel with the ribs* (Fig. 63) *and not perpendicular to them, as is often done.* It is obvious that when the plexímeter finger is placed vertically on the chest we obtain mixed resonance, because the stroke brings both healthy and diseased lung into vibration in cases of limited lesions. *Only intercostal spaces should be percussed* because percussion of the ribs, which in themselves are to be considered as long plexímeters, brings out resonance due to vibrations of large areas of lung tissue which lie laterally, and not only from beneath the spot which we intend to strike at the given moment.

The usual way of beginning percussion at the top of the chest and

going gradually downward to the base has many disadvantages. It is much better to percuss from below upward. N. K. Wood[1] summarizes the reasons for this procedure as follows: "It is much easier for the ear to pick up a higher note from a lower than it is to do the reverse; it requires a much lighter stroke to bring out the normal note than the pathological; it is the rational plan to work from the normal as a standard toward the pathological. The reverse leads to faulty standards. The apices, as is well known, are most frequently affected and more rarely give a normal note. To start at the apex, therefore, is usually to commence with a pathological note. This prejudices the further examination. With downward percussion, the

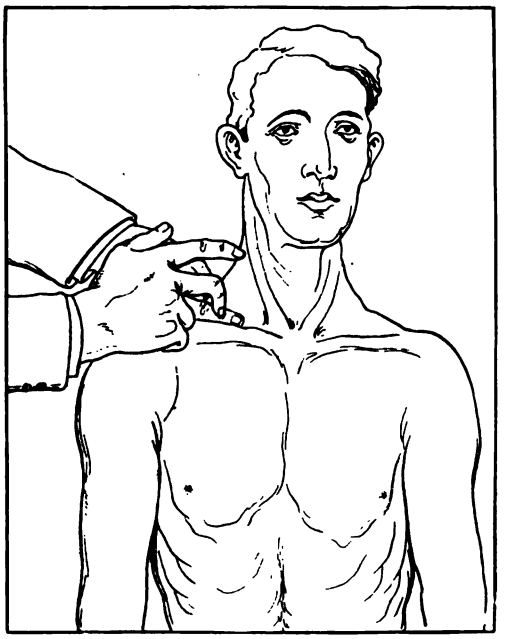


FIG. 63.—Percussion of the right apex.

higher note merges into the lower too imperceptibly to do accurate work. This is so for two reasons: (1) the mind becomes prejudiced in favor of a pathological note and consequently does not attempt to make fine distinctions, (2) a heavier stroke is required for the pathological note, and when the more resonant is reached the percussion is continued too heavily to detect what should be readily appreciated differences in the force of stroke necessary to bring out a good note. In this way the examiner deprives himself of a very important guide to collect accurate data."

[1] Jour. Am. Med. Assn., 1914, **63**, 1378.

The Hooked-finger Pleximeter.—In incipient phthisis we aim at localizing the smallest possible area of dulness, and at times the pleximeter finger is too large for the purpose. Plesch[1] has suggested that the pleximeter finger be flexed at the second phalanx to a right angle; the pulp only is applied to the chest and the distal end of the first phalanx is percussed (Fig. 64). This maneuver also enables the delimitation of the boundaries of the apex, or the determination of the condition of the apex behind the heads of the sternocleidomastoid, which is often of great importance.

Position of the Patient.—The patient should sit on a revolving stool, or better stand up with his head in the middle line, arms hanging by the side in a relaxed condition (Fig. 63). Contraction of any of the muscles of the chest on one side may greatly interfere with the results. When the back is percussed the patient is asked to fold his arms each on the opposite shoulder with a view to removing the scapulæ as far outward as possible. With these bones in the normal position the

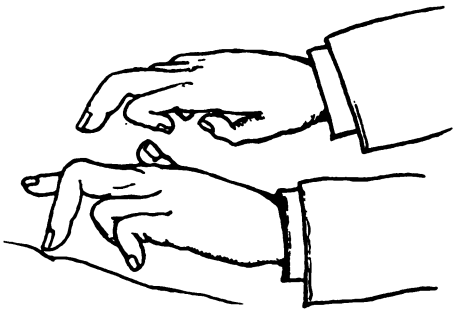


FIG. 64.—Hooked-finger percussion.

greater part of the lung in the supraspinous fossæ is beneath the bony thorax, and the apex is partly covered by the shoulder-blades. To hammer away in the supraspinous fossæ, as we often see done, is a waste of time and energy, because percussion there strikes bone and thick muscles, and the waves hardly, if at all, penetrate into the lung. But with folded arms, each over the opposite shoulder, or the patient embracing the back of a chair, the shoulder-blades are moved far away from the median line of the body, thus exposing the lung covered by comparatively thin parietes.

When it is desired to bring out the finer shades of resonance or, in doubtful cases, it is advisable to have the patient lying down on an upholstered couch or an examining table. Placing the patient with his back near a wall or door may help in bringing out points which might otherwise escape attention.

Comparative Percussion.—When percussing, we compare symmetrically corresponding areas on both sides of the chest and percuss

[1] München. med. Wchnschr., 1902, **49**, 620.

with equal force while striking each side. This is especially important because there is no standard resonance for a healthy chest; every individual has his own resonance which depends on many factors, mainly the vibration of the chest walls and the contents of the thoracic cavity, which are inconstant values. But in the normal chest the resonance, in all its properties such as duration, intensity, quality and pitch, are practically the same on both sides.

In incipient cases there are "seats of election"—points where dulness is most likely to be encountered if there is an apical lesion. Anteriorly, it is mostly under the inner third of the clavicle, and posteriorly at the inner margin of the upper half of the scapula.

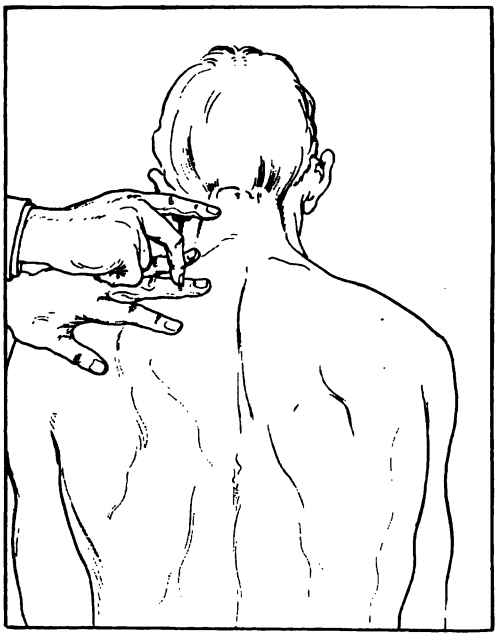


FIG. 65.—Percussion of the left apex posteriorly.

A small area of defective resonance can often be discovered by immediate percussion directly over the clavicle, comparing one side with the other. Immediately above and below the clavicle mediate percussion will bring it out, if it is present. If, on light percussion, impairment of resonance is discovered, the force of the blow is diminished to a minimum, thus delimiting the affected area, and we can again percuss the same spot, gradually increasing the force of the blow, always having in mind the thickness of the integuments, with a view to ascertaining the degree of dulness. If the dulness disappears with a heavy stroke, the lesion is of slight extent and superficial, or there

may be a thickened pleura; but if it persists, we may feel confident that we are dealing with an extensive area of airless tissue.

Posteriorly, we look for dulness over the apices of the upper and lower lobes of the lung. The former is located in the supraspinous fossa near the spine and reaches the first thoracic spine; the latter is lower in the right side, reaches the fourth thoracic spine and higher in the left side at the third thoracic spine (Fig. 60). If impairment of resonance is present in incipient cases, it will be found at one of these four points.

While doing comparative percussion of apices it is imperative to remember that, in the majority of healthy persons, the resonance over

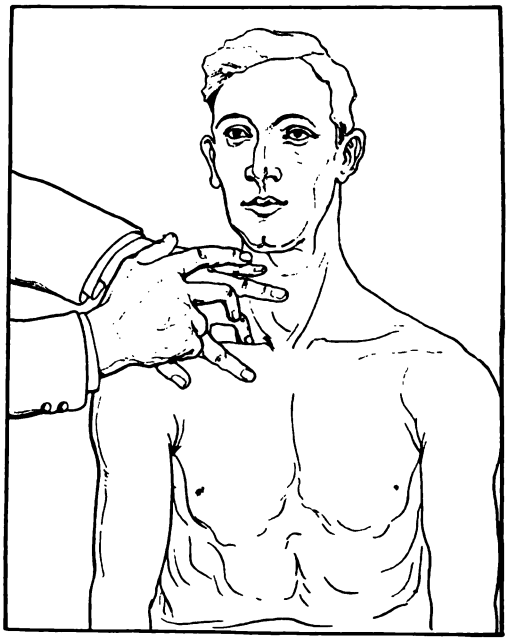


FIG. 66.—Hooked-finger percussion of the apex.

the right apex above the third rib is somewhat defective; the note is shorter and of higher pitch. This has been attributed to various causes. Investigations of George Fetterolf and George W. Norris[1] have shown that it is due to the anterior position of the large vessels in relation to the right apex, as compared with the left; to the consequent encroachment upon, and reduction in size of, the right apex and to the contact of the inner surface of the right apex with the resonating trachea, while the left is in contact with non-resonating solid tissue. In right-sided lesions, when the signs are inconclusive, topographical percussion is therefore best.

[1] Am. Jour. Med. Sci., 1912, **143**, 637.

Tympanitic Resonance in Incipient Lesions.—In the early stages the absence of distinct dulness in any part of the thorax is not always an indication of the absence of tuberculous infiltration. Impairment of resonance can only be brought out when the focus is at least one inch in diameter, although some, like Flint and Oestreich, are said to have detected smaller foci. But small disseminated tubercles, before they become confluent, may alter the resonance in an altogether different direction. Causing relaxation or hyperfunction of the surrounding lung tissue, they impart a tympanitic note on percussion. This tympany is of great importance in the diagnosis of incipient lesions, and is usually the cause why two competent observers will at times detect the lesion on different sides of the chest.

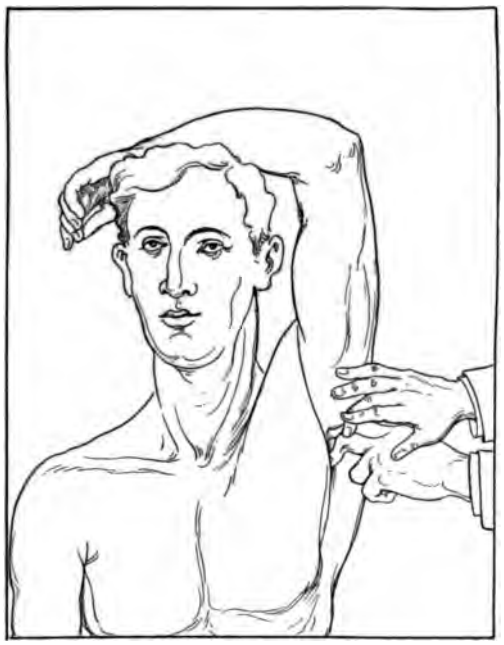


FIG. 67.—Percussion of the axilla.

Everyone who has had the opportunity and inclination to watch incipient tuberculous lesions has met with cases in which the first sign obtained on percussion is localized tympany, which subsequently changes into dulness with a tympanitic overnote, and finally becomes dull. Tympany in one supraspinous fossa, when accompanied by suspicious symptoms, is to be taken seriously; it may be the sole indication of small disseminated tubercles.

Absence of percussion signs, on the other hand, does not exclude incipient phthisis, because the lesion may be located deeply, subapically, or centrally, or it may be altogether a more malignant process—

miliary, or disseminated, tubercles all over the lungs which have not yet become confluent. In the same manner, extensive tympany over one lobe, or one lung, with fever, cough, etc., may be an indication of extensive tuberculization of the affected part. The outlook is not so good as when the tubercles are localized in a limited area.

Respiratory Percussion.—In doubtful cases it is advisable to study the changes in the resonance during extreme and held inspiration and expiration, as was suggested by J. M. Da Costa[1] half a century ago. He showed that "at the apices, and especially in the infraclavicular region, in the supraspinous fossæ, and on a line toward the spine, a

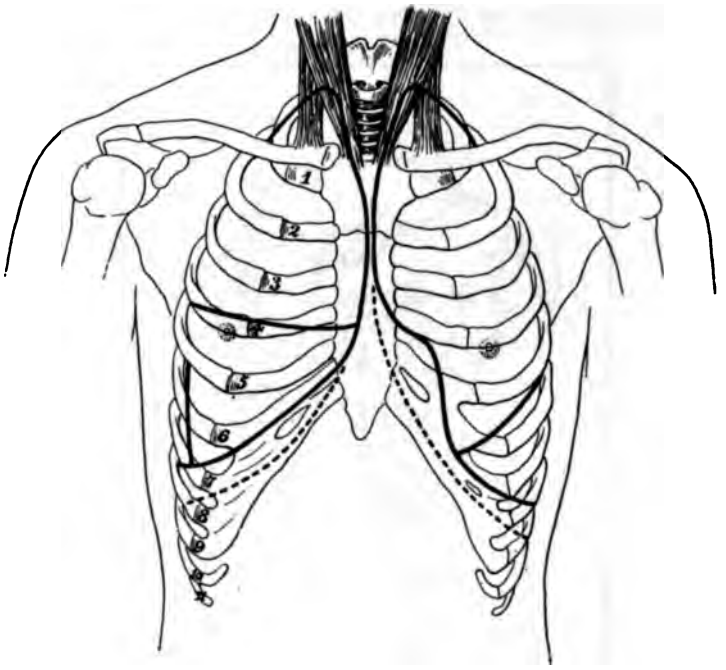


FIG. 68.—Lung margins according to Goldscheider.

full-held inspiration increases the resonance, makes the sound fuller and raises the pitch; and where, as is so common, the left side has normally a higher pitch, this disparity is preserved." A held and complete expiration will greatly lessen the resonance and lower the pitch at the apices. "In the held inspiration we obtain a greater mass of tone; in held expiration, the reverse." This change of resonance was found by Da Costa to remain unaffected in bronchitis; but in phthisis, even in the earlier stages, the affected area shows the reverse —a long-held inspiration gives a duller note than that observed on the healthy side.

[1] Am. Jour. Med. Sci., 1875, **70**, 17.

This change of note during held inspiration and expiration is brought out very clearly by light percussion and is of great value in doubtful cases. When the infiltration increases in extent, involving the larger part of the apical parenchyma, the dulness on percussion is no longer modified by the forced and held expiration and inspiration. Hence we have in this method a very good test as to the extent of involvement in the tuberculous process. Aufrecht[1] confirmed these findings.

Topographical Percussion of the Pulmonary Apices.—There are cases of incipient phthisis in which comparative percussion gives no conclusive information, and only topographical percussion—mapping

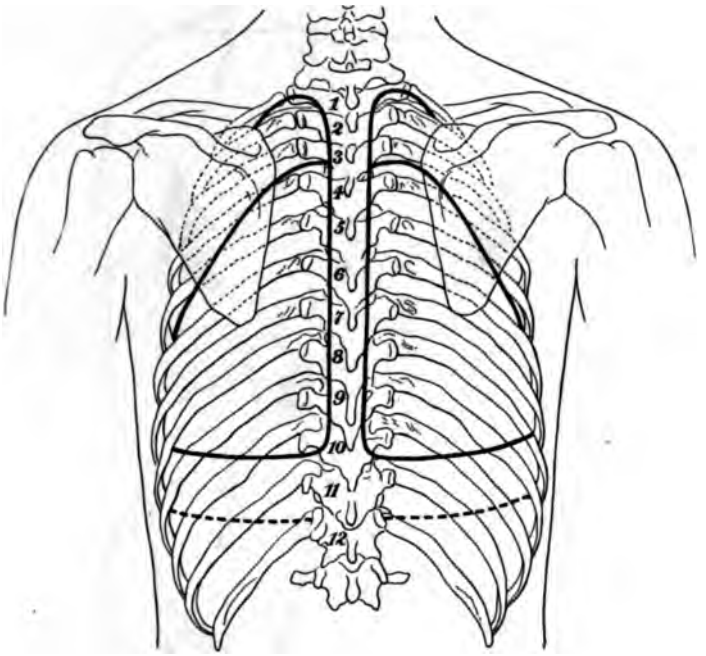


FIG. 69.—Lung margins according to Goldscheider.

out the limits of the apical resonance—may clear up the case. This can only be done intelligently when we have clear ideas as to the limits of these resonant areas in the healthy person.

Krönig[2] showed that the resonant areas project as cones anteriorly and posteriorly, and that these two cones are united on the top of the shoulders by a narrow strip of resonance—*the isthmus* (Figs. 70 and 71). With careful and very light percussion we can easily map out the mesial line which runs in front, beginning at the sternoclavicular articulation, upward and outward forming a concavity inward, while

[1] Berl. klin. Wchnschr., 1912, **49**, 101.
Deutsch. Klinik, 1907, **11**, 581 and 634.

posteriorly the line forms a convexity and ends at the level of the lower border of the second thoracic spinous process. The external line separating the resonant apex from the dull shoulder and neck runs from the middle of the anterior border of the trapezius, curving downward and reaching the clavicle at the junction of the middle and outer thirds and continuing obliquely downward toward the axilla; proceeding upward, it forms a convexity toward the neck, crossing the shoulders,

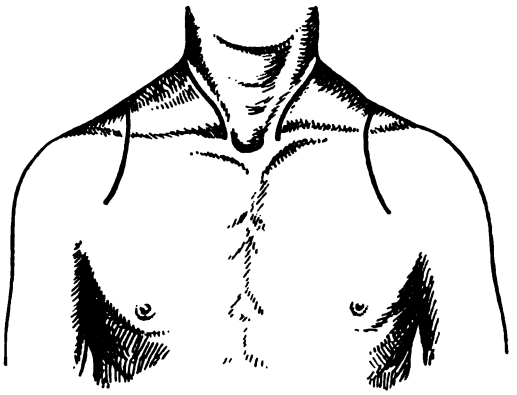


FIG. 70

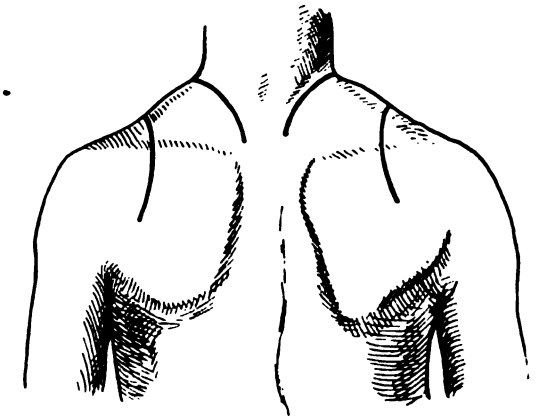


FIG. 71

FIGS. 70 and 71.—Krönig's apical resonant areas.

on the top of which it is separated from the mesial line by a resonant space of about 2 or 3 cm. forming the isthmus, and proceeding downward with its concavity outward, terminating a couple of centimeters outside of the middle line of the scapula. Normally the height of the apex is anteriorly about 2 or 3 cm. above the clavicle, and posteriorly, on a level with the first thoracic spine, about 2 cm. outside of the middle line of the body.

It is important to remember that the pleximeter finger should be applied parallel with the line we expect to delineate; in this case at right angles with the clavicle. It is better to percuss from the lower parts of the chest upward, because in the former the normal note is usually found in early cases and it is always best to compare normal resonance with defective by striking the former first, as was already indicated.

Changes in Apical Resonance in Phthisis.—When the resonant areas are marked out on the chest of a healthy person, their height and width are practically the same on both sides. But in phthisis one side will be found contracted. Recalling that a tuberculous lesion in

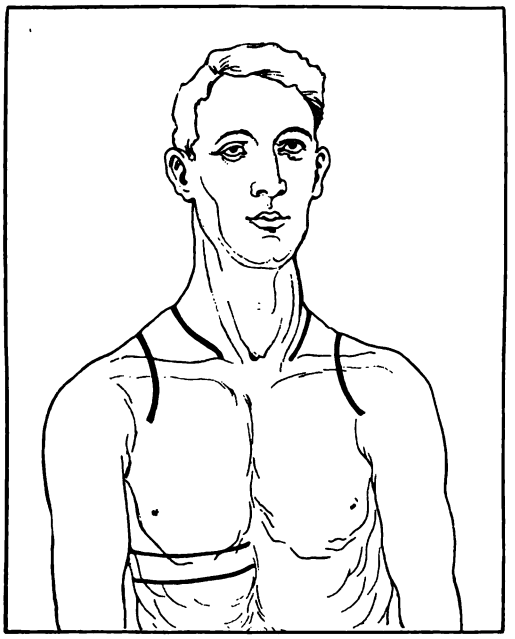


FIG. 72.—Contraction of the resonant area of the left apex.

the apex involves shrinkage of the pulmonary parenchyma, we have an explanation for this phenomenon. The extent of the shrinkage depends on many factors, mainly the degree of pulmonary retraction and the location of the lesion. When the lesion is centrally located, shrinkage of the apex is greater than when it is located at the periphery or under the pleura, as has been shown by Oestreich, obviously because in the former case traction is exerted on all sides. Autopsy findings show conclusively that this shrinkage occurs quite early, much earlier than is generally appreciated, and for this reason we may get a clear view as to the condition of the lung in that region, by percussing the apices and mapping out Krönig's resonant areas.

Shrinkage manifests itself in two ways:

1. By a narrowing of the field of resonance on the affected side. This can be established by actual measurement. The isthmus in healthy persons is about 2 or 3 cm. in width, and when we find it less than 1.5 cm. in width, it requires investigation. The width of the base of the resonant cone may be measured simply in finger-breadths, as has been recommended by R. N. Philip.[1] Both sides are to be of the same width.

2. By a blurring of the line separating the resonant from the dull parts (Figs. 73 and 74). While in health we can easily percuss out a

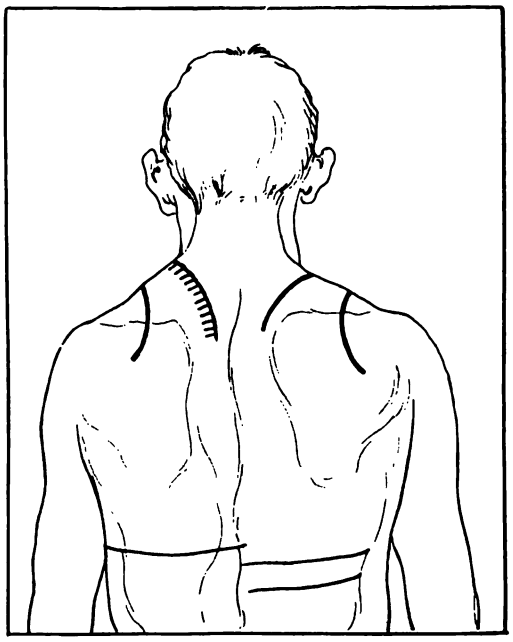


FIG. 73.—Krönig's resonant areas, showing a band of doubtful or relative resonance at the mesial border of the left apex; also retraction of the lower margin of the left lung.

clear line of demarcation, in tuberculous apices there is often an interval in which the resonance is doubtful. This is mostly found at the inner outlines, but may be found at both sides. Krönig attributed it to changes in the tension of apical parenchyma at the margin of the affected parts. These points are better illustrated than described (Fig. 73), and in practice after the outlines of the apices have been marked out with a skin pencil, any existing differences in the outlines of the apices when one side is compared with the other are noted at a glance and need no measuring.

[1] Edinburgh Med. Jour., 1907, **22**, 473.

Sources of Error.—Krönig's method is of excellent service in most cases of incipient phthisis. But we often meet with cases in which after careful and time-consuming work, the results attained are unsatisfactory. I have seen cases of phthisis in which no dislocation of any of the outlines of the apical resonance could be made out. Then there are numerous cases in which contraction of the apex is made out very nicely, but there is no active phthisis. This is especially true of "collapse induration," which will be discussed later on. Healed tuberculous lesions also leave contracted apices and what we seek to determine is the presence of active phthisis. Walter C. Klotz found differences in the two sides very frequent in non-tuberculous indi-

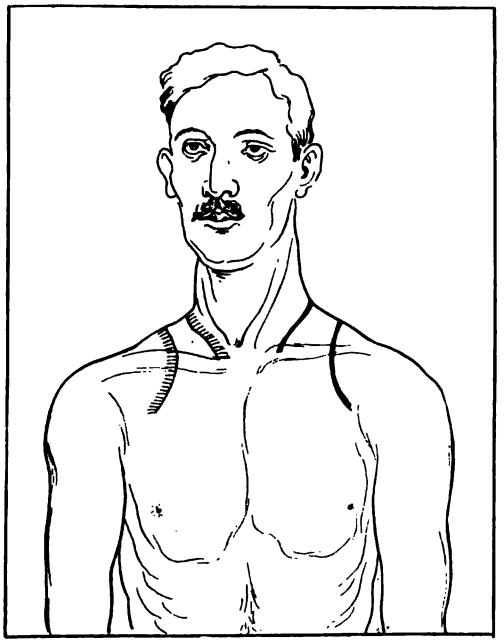


FIG. 74.—Bands of doubtful resonance on both sides of the right apex anteriorly.

viduals; the right side is often narrower, regardless of the site of the most extensive lesion. His conclusion, which is in agreement with our experience, is to the effect that unless the disparity of the apical percussion field, expressed in terms of Krönig's isthmus, is very marked, it does not necessarily point toward tuberculosis of the corresponding side. Such a disparity is also of less significance on the right side than on the left.

Krönig stated that in phthisis the motion of the base is invariably affected at an early stage, while in non-tuberculous apical lesions the expansion of the lower margins of the lung remains normal. This does not hold in practice. There are many cases of phthisis in which

the base retains its normal mobility during inspiration and expiration, and the reverse. The reason for the occasional failure of this method of percussion lies in the fact that the resonant area is not an outline of the true anatomical apex, but merely a projection of the same lung tissue in various directions (Figs. 77 and 78). The fact is that it is impossible to project the top of the lung on the surface of the body, considering its peculiar anatomical position and form. Krönig's isthmus, for instance, does not exist at all, and we must remember that only the mesial border corresponds to the anatomical margin of the lung anteriorly and posteriorly. The lateral border cannot be

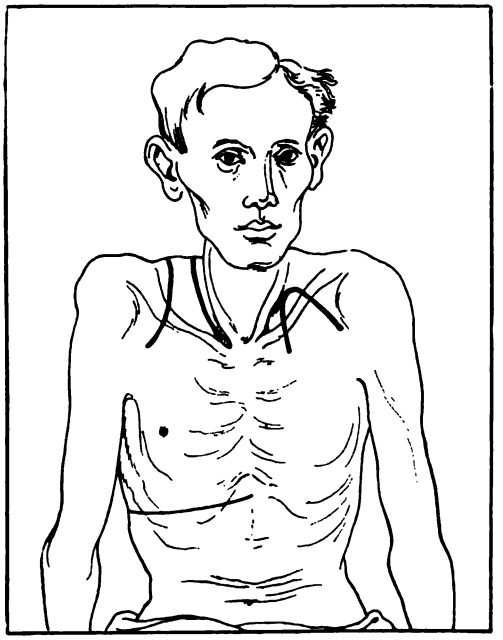


FIG. 75.—Frequent findings with Krönig's method of percussion in advanced cases. Retraction of the left lung.

determined with exactness in most cases because the percussion wave strikes the spot tangentially. In patients with marked scoliosis, the method is of no value at all.

Goldscheider's Method of Apical Percussion.—Anatomical studies by Goldscheider,[1] as well as orthodiagraphic examination of the lungs in their relation to the bony thorax, show conclusively that there is no lung tissue in most of the resonant area percussed out by Krönig's method. Anteriorly, the apex lies beneath the two heads of the sternocleidomastoid, protruding above the inner third of the clavicle for

[1] Berl. klin. Wchnschr., 1907, **40**, 1267 and 1309.

about one inch in height. This is seen clinically when emaciated persons cough and the lung is blown up above the clavicle, or in wasted infants during crying spells. Posteriorly, the apex of the lung lies close to the spinal column, reaching as high as the spinous process of the first thoracic vertebra. But there it is impossible to obtain resonance from it because it is covered by a bony transverse process, rib and thick muscles.

Goldscheider,[1] for these anatomical reasons, devised another method of obtaining the resonance of the true anatomical apex, which we discussed in detail elsewhere.[2] From the complicated procedure of Goldscheider all that is of utility in doubtful cases is the determination

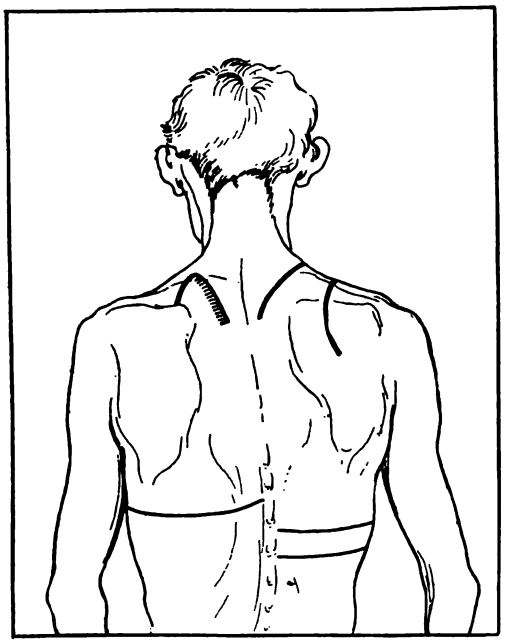


FIG. 76.—Same patient as in Fig. 75; findings posteriorly.

of the height of the apex between the heads of the sternocleidomastoid, which can easily be done by percussing from below upward with the hooked finger as a plexímeter and comparing the two sides. Posteriorly, the lung resonance should reach the tip of the spinous process of the first thoracic vertebra on both sides. The height of the apices on both sides normally should be the same, and if it is found shorter on one side, it demands investigation as to the cause. In connection with other symptoms, it is strongly in favor of tuberculosis. But here

[1] Ztschr. f. klin. Med., 1910, **69**, 205.
[2] New York Med. Jour., 1913, **97**, 799.

again, it may be an old, healed lesion. The distinction between active and healed lesions is made by means other than percussion.

Tidal Percussion.—After ascertaining the limits of the apices, the base is to be delineated with a view of determining the vertical movements of the lung in the pleural sinus during both phases of respiration. This gives us information as to the presence or absence of emphysema, especially in fibroid phthisis, pleural adhesions, which are of such immense interest when contemplating the application of a therapeutic pneumothorax, etc.

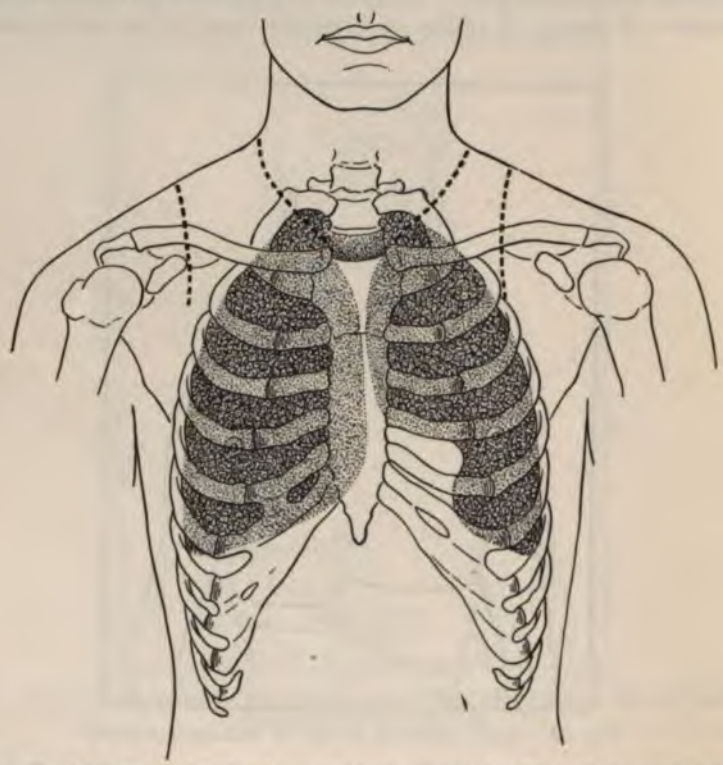


FIG. 77.—Showing that Krönig's resonant areas are not outlines of the apical margins but are merely projections of the same lung tissue in various directions. (After Goldscheider.)

The lower margins of the lung resonance are first ascertained by percussion while the patient breathes normally and quietly, and marked with a dermographic pencil. Then the patient is directed to take a deep breath, and hold it as long as possible, while we again percuss and ascertain the lower limits of the lung, and again mark them with the pencil. In healthy persons the difference in these two lines is between one and two and a half inches. It is to be borne in mind that on the left side the lung margin is naturally about an inch lower

than on the right; also that the expansion is greater in the axillary line anteriorly than posteriorly. In emphysematous subjects, also in the senile, and in those with deformed chests, expansion may be very little or *nil*. Pain while breathing may have the same effect. On the left side, when there is no expansion anteriorly at Traube's semilunar space, it is an indication of pleural adhesions, or effusion; an increase in the tympany at that space indicates retraction of the left lung, not infrequent in phthisis.

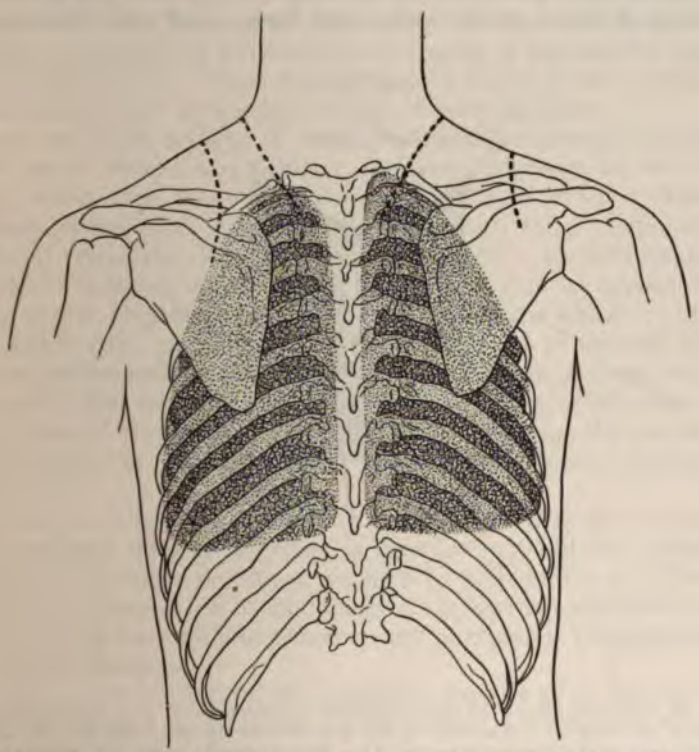


FIG. 78.—Showing that Krönig's resonant areas are not outlines of the apical margins, but are merely projections of the same lung tissue in various directions. In the supraspinous fossæ there is no lung tissue at all. (After Goldscheider.)

In most cases of incipient phthisis the respiratory excursion of the affected lung is more or less restricted, and when there are adhesions, there is unilateral absence of respiratory excursions. But since we have been interested in pleural adhesions while making artificial pneumothorax, we find that these signs are not absolutely reliable.

Percussion in Advanced Phthisis.—With the advance of the disease the percussion findings become more and more varied and scattered all over the chest, and the difficulties of determining the exact condition of the lungs from percussion findings alone, more and more unsur-

mountable. The dulness elicited is usually due not only to the active lesions, but also to such as have healed or are quiescent; to thickened pleura, which is usually a conservative process; to pleural effusions, displacements of the heart, diaphragm, liver, stomach, etc. Some of these processes are permanent, others appear for a short time and disappear. Localized emphysema, transient or permanent, due to vicarious function, often obscures deeply lying airless tissue.

In most cases, however, we find that one lung shows dense dulness in its upper part, usually as far as the third or fourth rib, as well as retraction of one or, more rarely, both bases. But even this may be

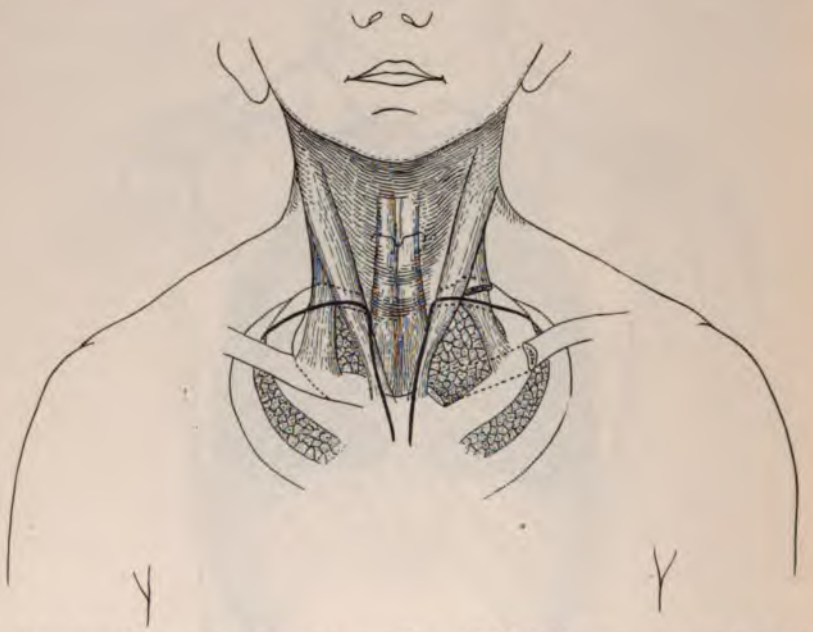


FIG. 79.—Topography of the apex according to Goldscheider: —— upper and mesial borders of the lung; - - - - - borders of the first rib and clavicle. On the left side the clavicular head of the sternocleidomastoid has been removed so that the scalenus anticus is visible. The upper border of the lung is somewhat higher than the first rib.

due to healed or quiescent old lesions. We also find a frequent area of dulness in one and, at times, in both interscapular spaces due to lesions of the apices of the lower lobes, or enlarged glands. At times the dulness runs along the lines of the interlobar fissures anteriorly and posteriorly. To map out such areas of dulness may be of scientific interest, but the diagnosis of these cases rests on other methods of exploration, especially the subjective symptoms. Signs of excavation are discussed elsewhere. (See Chapter XX.)

Sources of Error in Signs Elicited by Percussion.—When finding defective resonance over one apex, contraction of Krönig's resonant area on one side, or one apex shorter than the other, thus indicating

pulmonary retraction, are we justified in considering the patient sick with active phthisis? Are differences in resonance elicited when the two sides of the chest are symmetrically and comparatively percussed, especially in its upper third, sure indications of active phthisis?

These problems confront the clinician quite often, and they can only be answered by an intelligent consideration of the causes of defective resonance and dulness, which are mainly airless lung tissue, and which may be due to many causes in addition to tuberculosis. Besides, we may have differences in the resonance due to faulty technic in percussion, also because of asymmetry of the chest in cases of kyphosis or scoliosis, or unilateral hypertrophy of the muscles due to occupational effects. These facts are to be borne in mind while we attempt to interpret percussion findings in early phthisis.

There are other sources of error. Chronic pneumonic processes, healed apical lesions and pleurisy are very common, as we have already shown, and many leave some airless tissue which is detected by careful percussion. So that even if due to tuberculosis, apical dulness or retraction does not always mean active phthisis requiring therapeutic intervention. Collapse induration, due to inhalation of dust in mouth-breathers, may show percussion signs which are undistinguishable from phthisis, if we should rely on percussion alone. We also occasionally find dulness in the apices in persons leading a sedentary life, and who do not breathe deeply, especially chlorotic girls. Some of these cases are cleared up by directing the patient to breathe deeply for some minutes, or practising Da Costa's respiratory percussion.

We also meet now and then with persons in whom the resonance on one or both sides of the chest is defective without any excessive adiposity or strongly developed muscles to account for it. The air content of the lungs is less in childhood than in later life, and it decreases with old age, often without showing any anatomical changes in the lungs at the autopsy.

In many cases a study of the overlying muscles as to rigidity and atrophy has helped me immensely, while in others it was of no avail.

Diagnostic Value of Percussion.—In cases presenting symptoms of phthisis such as fever, cough, nightsweats, etc., percussion findings alone are often sufficient to localize the lesion, and in many cases it will be found by prolonged observation that a lesion develops in the apex where we originally found only defective resonance or contraction of the field of resonance, though auscultatory signs were wanting.

Percussion findings alone, without any general symptoms of phthisis, are not conclusive, just as in roentgenography a shadow over an apex does not prove an active tuberculous lesion. It is only in connection with constitutional symptoms that percussion, like any other single sign, can be utilized for diagnosis.

However, whenever found, defective resonance in an apex demands careful investigation and watching of the case until a reason is found for its existence.

CHAPTER XVI.

AUSCULTATION OF THE CHEST IN PHTHISIS.

WE have shown that percussion is a most valuable diagnostic method in early phthisis, even more valuable than in the later stages, and will often give definite information as to the air content of the lungs much earlier than other methods. Auscultation is just as valuable for other reasons. At times it affords information in cases in which the lesion is centrally located, and in tuberculosis grafted on an emphysematous lung, when percussion and even roentgenography may fail. Similarly, in advanced cases where the lesion is extending, altered breath sounds and rales may often be found in advance of dulness. On the other hand, acute cases, especially miliary tuberculosis, may show normal breath sounds and no rales, and in chronic cases with deeply lying cavities the normal lung tissue conceals the signs of excavation. In the former diffuse tympany, while in the latter percussion or roentgenography, may disclose the exact state of affairs.

Believing that the technic of auscultation is much easier to master than that of percussion, many have discarded the latter and rely solely on the former, which is a grave error. The fact is that *it is just as difficult to acquire skill in proper auscultation of the chest, and in interpreting the findings correctly, as to percuss properly.* Some, like Goldscheider[1] and Clive Riviere,[2] believe that auscultation is even more difficult to master. It is because of faulty technic that auscultation does not yield all the information that can be obtained by this method.

Technic of Auscultation.—The patient should be stripped to the waist, just as for percussion, and seated on a high revolving stool, so as to be accessible from all sides. Before beginning auscultation the physician must assure himself that the patient knows how to breathe properly and if not, which is very often the case, proper instruction is to be given objectively. One important drawback to auscultation is that many patients do not know how to exhale—they just inspire jerkily, and stop with inflated chests. Others, usually such as have led a sedentary life and never expanded their chests properly; inhale and exhale quickly in rapid succession so that it is difficult to follow each phase of respiration. While in the vast majority a little instruction suffices, at times we meet with some, and not exclusively among those reputed to be ignorant, who will not breathe properly for our purposes, especially nervous individuals, and the

[1] Ztschr. f. klin. Medizin., 1910, **69**, 205.
[2] Early Diagnosis of Tubercle, London, 1914, p. 22.

examination must be postponed till they become accustomed to the physician.

The breathing must be regular, rhythmic, somewhat deeper than usual, and preferably through the nose, because when the air enters this way the lungs expand much better and more uniformly. Mouth-breathing occasionally induces cough. In cases of nasal obstruction the patient breathes through his mouth, but we must guard against noises arising in the pharynx, especially those created by the soft palate, which impart a bronchial or blowing character to the breath sounds and, at times, give an impression of prolonged expiratory murmur, when in fact there is nothing of the kind.

Special attention should be paid to expiration, during which the patient should empty his chest as much as possible, without any undue exertion, and that each expiration should promptly be followed by a deep inspiration.

Any stethoscope to which the physician is accustomed may be used. The writer prefers the Bowles model, and the one devised by J. J. Singer, of St. Louis, has given satisfaction. The bell should be applied carefully in the intercostal spaces, especially in emaciated persons, so that it makes an air-tight connection with the skin. It should be held firmly but without any undue pressure, thus excluding all extraneous noises. Movement of the bell of the stethoscope upon the surface of the body interferes greatly with proper auscultation and should be avoided.

Single Phase Auscultation.—To appreciate slight changes in the duration and quality of the respiratory murmur it is important to listen to each phase of the respiratory act separately. Grancher's[1] method has served me best. It consists in first listening to the inspiratory murmur and to neglect at the time the expiratory murmur; and when listening to the latter the former is to be neglected. Rales are always looked for separately, after we have a clear idea as to the character of the breath sounds.

Beginning, for instance, with auscultation of the left apex, we listen attentively to the inspiratory murmur, and while the patient exhales, the bell of the stethoscope is quickly carried over to a corresponding point on the right side of the chest, and we listen to an inspiration. The inspiratory murmur is thus compared right and left, and any differences that may be found are carefully noted. In this manner the slightest change in the murmur on one side can be best appreciated, because we have a standard in the unaffected side. Only when both sides of the chest are affected is this method uninstructive, because we do not have an immediate impression of a normal inspiratory murmur. The expiratory murmur is to be studied in the same manner, carrying over the bell of the stethoscope while the patient inspires, and noting the difference. While listening to these

[1] Maladies de l'appareil respiratoire, Paris, 1890.

murmurs, no attention at all is paid to any adventitious sounds which may be present. These are left for separate study.

This method of auscultation, devised by Grancher, and hardly ever mentioned in our text-books, is the only one that can bring out all the changes in the respiratory murmur heard in really incipient pulmonary lesions, and should be used exclusively.

The Normal Respiratory Murmurs.—The most important prerequisite of proper interpretation of auscultatory findings in pathological conditions of the lungs is a knowledge of, and experience with, the respiratory murmur audible in normal chests. Without this knowledge we cannot expect to appreciate slight changes audible during either phase of the respiratory act in early phthisis. It is because of the disregard of the qualities of the physiological breath sounds that slight changes are overlooked, and many state that only with the appearance of adventitious sounds can a positive diagnosis be made, which is decidedly wrong, just as is waiting for tubercle bacilli to make their appearance in the sputum. One who wants to appreciate the early changes of phthisis cannot auscultate normal chests too often.

The physiological, or vesicular, respiratory murmur shows that the pulmonary parenchyma at the auscultated area contains air which enters with each act of inspiration and leaves with each act of expiration without meeting any obstruction in its course. During inspiration it is audible with different degrees of intensity all over the chest as a sighing, whispering rustle; during expiration there is either no murmur at all, or, more commonly, a very faint noise is heard which is somewhat lower pitched than, and it lasts but one-fifth the time of, the inspiratory murmur, notwithstanding that expiration actually lasts longer than inspiration.

Without entering into the problem of the origin of these murmurs, whether they are produced in the glottis or in the air cells in the areas under examination, we want to emphasize that it is important to bear in mind while auscultating that any changes in pitch, quality and rhythm noted during either phase of respiration are to be given careful attention in cases in which early phthisis is suspected.

Feeble Breathing.—When meeting a patient with a really incipient lesion, which is not often our privilege because when they present themselves the lesion is usually more advanced than is generally appreciated, we find no adventitious sounds, no changes in the type of breathing, no bronchovesicular or bronchial breathing, etc. The most common change in the breath sounds at this stage is feeble breathing, or, more rarely, complete absence of the respiratory murmur over a circumscribed area in one of the apices, mostly found posteriorly near or above the spine of the scapula, the *zone d'alarme* of some French authors,[1] and anteriorly beneath the inner third of the clavicle. At times this feeble murmur is blowing or even bronchial in

[1] Sergent, Le Monde Médical, 1912, **22**, 1121; La Clinique, 1913, **8**, 437.

character and at the end of inspiration some dry crackling may be heard.

It is noteworthy that while very few modern authors mention feeble breath sounds in incipient tuberculosis, the great French clinician of the first half of the nineteenth century, Andral, already considered it a good and reliable sign. He says: "We have ascertained weakness of the respiratory murmur, or even its total absence, in points where, after death, we found tubercles scattered in greater or less number in the midst of the pulmonary parenchyma very much indurated, and became entirely impermeable to the air."

To be of diagnostic significance this feeble breathing must be localized over one apex, circumscribed, fixed and persistent for some time, and uninfluenced by respiratory efforts and cough. It is an indication of peribronchial tuberculous infiltration compressing some bronchioles, thus creating atelectasis of the alveoli they supply; or of localized pleurisy interfering with the respiratory activity of the alveoli in the affected area.

"In massive caseation," says Colonel Bushnell, "the tissues have lost their elasticity and, in so far as they are caseated, do not expand at all in inspiration. Ordinary breath sounds are absent in such cases, or are present enfeebled in less complete caseations. Ordinarily what is heard is a weak and distant bronchial breathing, conducted from the deep bronchi and mingled with the coarse rales characteristic of these tubes."

Localized feeble breath sounds are also found over healed tuberculous lesions, or adhesions of the apical pleura following abortive tuberculosis. But during the early stage of active phthisis feeble breathing is accompanied by constitutional symptoms, such as cough, fever, tachycardia, etc., and usually some signs are elicited by percussion of the same area. As Bezançon[1] has pointed out, *in the absence of constitutional symptoms, feeble breathing at one apex is a sign of a healed tuberculous lesion.*

In advanced phthisis, we very often meet with limited areas of feeble or absent breathing, but vigorous cough removes the plug which obstructs the entry of air into a bronchus and breath sounds are again audible. It is noteworthy and of diagnostic importance that atelectasis is frequently produced by plugging of a bronchus and the resulting resorption of the air from the alveoli may produce dulness over the area supplied by that bronchus, but no breath sounds, no adventitious sounds are heard. Occurring at the base, it is often difficult to distinguish it from thickened or adherent pleura, which is also characterized by feeble or absent breathing, as is pleural exudate. Likewise, over old bronchiectatic cavities, the breath sounds are often lacking or are feeble, though the rales are quite pronounced, consonating.

In acute pneumonic phthisis I have repeatedly met feeble breath

[1] Rev. de la tuberculose, 1913, **10**, 1.

sounds in addition to dulness elicited over the affected lobe of the lung; at times there was even absence of all breath murmurs, but some moist subcrepitant rales were audible over the same region. Similarly, we may meet, during febrile exacerbations in advanced cases, feeble breathing over newly affected areas, which later changes into bronchial breathing, etc.

Rough or Granular Breathing.—This is often found in incipient cases. Here again it is the inspiratory murmur that is especially affected. It is dry, rough and low-pitched. It should not be confounded with puerile or harsh breathing:. Granular breathing may be altogether diminished in intensity, or even very faint, while puerile breathing is always intense and emphatically pure. On the other hand, in granular breathing there is always a suspicion that adventitious sounds or noises are superadding the inspiratory murmur. According to Sahli, it is a sign of bronchial catarrh; there is either partial impermeability of the bronchi producing unequal respiratory excursions of the affected lung area, or else the accompanying noises are derived from the secretions causing partial stenosis or irregularity in the lumen. When these accompanying noises can be plainly isolated we call them rales, but as they remain indistinct and blended, the vesicular breathing becomes impure, granular or rough. It is generally heard over the supraspinous fossæ, or above and beneath the clavicle.

As has been pointed out by Bray,[1] for a satisfactory demonstration of granular breathing at the apex, the muscles of this region must be in a state of relaxaction, because their active contraction may at times produce sounds indistinguishable from granular breathing, and thus lead to confusion in diagnosis. He suggests to listen while the patient breathes abdominally. In this type of breathing, respiration is conducted solely by the piston-like excursions of the diaphragm, the thorax is fixed, and the muscles in the region of the apex relaxed. Only when granular breathing is heard while the patient breathes abdominally, thus eliminating extrapulmonary sounds, is one justified in concluding that the vesicular murmur has undergone a definite pathological alteration.

Grancher insists that granular breathing is a sure sign of incipient phthisis, and Clive Riviere speaks of it as the earliest auscultatory sign, while Piéry[2] says that it is nothing of the kind, but that it is a good sign of a cured lesion and due to cicatrization of a limited area of lung tissue, which is undoubtedly a fact. *I have seen many patients who presented granular breathing at an apex for years without showing any of the constitutional symptoms of phthisis.* On the other hand, I have full confidence in this sign when there are the usual general symptoms of phthisis, because I have repeatedly observed that in the very area first presenting feeble or granular breathing there subsequently developed typical lesions of phthisis. Of course, one must always bear

[1] Jour. Am. Med. Assn., 1917, **69**, 1762.
[2] La tuberculose pulmonaire, Paris, 1910, p. 311.

in mind that the absence of constitutional symptoms is an indication that the granular breathing is probably due to a cicatrix remaining after a tuberculous lesion has healed.

Interrupted or Cog-wheel Breathing.—The *respiration saccadée* of the French is another anomalous type of breath sounds which has for a long time been considered characteristic of early phthisis. The inspiratory murmur is not smooth and continuous, as in normal respiration, but is broken, so that it appears jerky, divided into several more or less distinct parts. It differs from rough breathing by the fact that each portion of the sound retains its smooth, rustling character. It is apparently caused by the obstacles met by the air current while entering the alveoli. The breath sounds may be increased or, more commonly, decreased in intensity.

I find cog-wheel respiration only rarely a sign of incipient phthisis and am inclined to agree with Piéry, who says that in the region of the apex it is always an indication of pleural adhesions which are often the remains of a healed tuberculous lesion. In some cases, however, it is met with in the beginning of active phthisis and the fact that in the later stages of the disease it can very often be heard along the borders of advancing lesions shows that the factors producing it may be of the first disturbances of the respiratory murmur in the areas of impaired breathing capacity around infiltrated portions of the lung.

Cog-wheel breathing is occasionally heard over chests in nervous patients, or such as have pains due to acute pleurisy, or who shiver during the examination. But then it is heard all over the chest, while in phthisis it is localized over a limited area.

Prolonged Expiration.—From what has been stated it is evident that *in the very early stages of phthisis, auscultation reveals only changes in the inspiratory murmur,* a point which cannot be too strongly emphasized. In older books on the subject we almost always read that changes in the expiratory murmur are pathognomonic of early phthisis, obviously because in former days incipient phthisis, as we know it today, was not recognized. In fact, because even today patients only rarely present themselves for examination at the very incipiency of the disease, we usually find a prolonged expiratory murmur at the first examination. But speaking as one who has had opportunities for examination of large numbers of persons who do not even suspect that they have any pulmonary trouble, and examining the lungs of everyone who comes under my care, I find that changes in the inspiratory murmur, such as feeble breath sounds, rough or cog-wheel breathing, are usually found earlier than changes in the expiratory murmur.

In normal vesicular breathing the expiratory murmur is either inaudible or, more commonly, it lasts only one-fifth to one-fourth the time of the inspiratory murmur. When it lasts as long as, or longer than, the inspiratory murmur it is undoubtedly pathological, though not necessarily of tuberculous origin. When audible all over the chest

it is an indication of bronchitis or pulmonary emphysema, but when we find it localized at one apex, its significance as a sign of phthisis is to be appreciated. It may be due to sclerosis of a limited portion of lung tissue, as is the case in healed tuberculous lesions. Indeed, when it also has a bronchial timber it is pathognomonic of this condition, and Turban speaks of it as "cicatricial respiration."

In active early lesions, a prolonged expiratory murmur, localized at an apex, is an indication of either catarrh of the smaller bronchioles, or pressure on these tubes, in cases in which infiltrations produce stenosis. It is therefore usually met with later than the changes in the inspiratory murmur, of which we spoke above. The prolonged expiratory murmur is often harsh and rough, and with the advance of the disease, it gradually acquires a bronchial character, finally becoming pure bronchial or tubular breathing. While we may meet it without any adventitious sounds, this is exceptional in my experience. On the other hand, it may be feeble and hardly audible and, at times, we hear the rales very clearly while the prolonged expiration is so feeble that it is only detected after careful listening.

There is another fact to be borne in mind while evaluating prolonged expiration as a sign of early phthisis. Not only may it be the sole indication of a healed lesion, as has already been stated, but in the right apex it may not be due to tuberculosis at all, especially in young adults with thin thoracic walls. In collapse induration it is not uncommon, while in persons working at dusty trades, such as stone-cutters, carpenters, miners, garment-workers, etc., the expiratory murmur at the right apex is very often harsh, rough and prolonged. Under the circumstances it is of more significance when found in the left apex, and in the right side a careful study of the constitutional symptoms must be made before attaching any diagnostic value to it.

Bronchial Breathing.—With the advance of the disease the disseminated tubercles in the lung conglomerate by growth and form a solid circumscribed mass, over which the breath sounds elicited on auscultation are more or less characteristic. The vesicular quality of the murmur changes by degrees, till it finally becomes high-pitched, clear and blowing during both inspiration and expiration, which is very prolonged.

Bronchial breathing is a sign of consolidation of lung tissue: The laryngotracheal murmur is transmitted and, according to Sahli, even magnified, while passing from the bronchi through consolidated lung tissue to the surface. It is thus heard over areas which are dull on percussion, particularly over the upper third of the chest anteriorly and posteriorly. During the course of chronic phthisis bronchial breathing is also caused by many complications which produce compression of the alveoli with resulting pulmonary atelectasis, as is the case in pleural effusions, pneumothorax, hydrothorax, etc. In these cases the bronchial breathing is engendered only when the alveoli and, at most, the bronchioles are compressed; when the large tubes are also obliterated by compression, no breath sounds at all are audible.

In acute phthisis, bronchial breathing is mainly caused by caseous infiltration of the affected areas, and it is harsher, louder and more high-pitched, the more compact and extensive the consolidation of lung tissue. Bronchial breathing in phthisis is not so loud and high-pitched as in pneumonia, and when it is encountered, it is an indication of an acute process which is probably progressive and of serious prognostic significance. It is therefore found early in the disease in acute pneumonic phthisis and during chronic phthisis over the seat of new extensions of the process, involving the larger part of a lobe, and in the terminal stages, when tuberculous bronchopneumonia complicates an old lesion and carries off the patient. In chronic phthisis, the higher the pitch of bronchial breathing, the greater the consolidation of lung tissue may be assumed.

It is a fact to be remembered that in the average case of chronic phthisis bronchial breathing does not appear suddenly but by slow degrees. The vesicular murmur is gradually transformed into bronchovesicular, which, with the subsequent consolidation of the process, finally becomes purely bronchial.

Bronchovesicular Breathing.—On rare occasions, we may find bronchial breathing with normal resonance over the same area; in fact, I have at times met it over areas emitting a tympanitic note on percussion, which is an indication that even small disseminated tubercles, which are incapable of producing dulness, but relax the lung tissue and cause tympany, may cause bronchial breathing.

But usually disseminated tubercles produce bronchovesicular breathing. We hear a mixture of both vesicular and bronchial sounds over the same area, the former originating in the small consolidated areas which transmit the laryngotracheal sounds, while the latter come from the alveoli of the unaffected lung tissue that surrounds the tubercles. It is thus clear that the presence of bronchovesicular breathing is an indication of small tubercles scattered within normal lung tissue. This is usually preceded by prolonged expiration, which changes by degrees into bronchovesicular breathing, and finally into bronchial, as has already been shown.

Bray, however, offers another ingenious explanation of the origin of bronchovesicular breathing. He points out that the lung glides beneath the ribs during respiration, and as a result the lung surface auscultated over any given area is considerably greater than that represented by the bell of the stethoscope. The area of lung auscultated will thus vary with the motion of the lung. Bronchovesicular breath sounds are heard when there are contiguous areas of consolidated and air containing lung tissue, which alternately pass across the auscultated field during respiration.

Sources of Error.—*Bronchial and bronchovesicular breathing per se are no indications of phthisis.* In addition to the many pathological conditions which may cause this type of breath sounds, we quite often hear it over healthy chests. There are many individuals in

whom bronchial breathing is heard all over the upper parts of the thorax. In the interscapular, right supraspinous and supraclavicular spaces it is very common in apparently healthy persons, especially during vigorous breathing. This is said by Bandelier and Röpke to be found in about one-third of healthy people; it is due to differences in the anatomical structure of the two apices. The right lung has three main bronchi, which favor the transmission of bronchial breathing more than the left, which has only two. It will be noted (Fig. 80) that the branches of the bronchus supplying the right apex are wider and go deeper into the parenchyma, thus affording better opportunities for transmission of sound to the surface, than in the left apex.

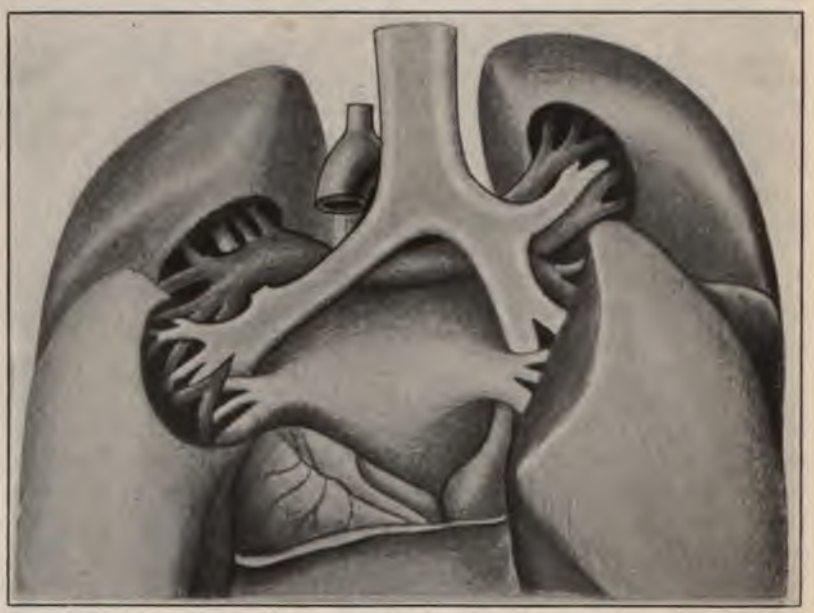


Fig. 80.—Topographical relations of the large bronchi and pulmonary bloodvessels after removal of the posterior wall of the thorax, the esophagus and descending aorta. Pulmonary artery dark; pulmonary vein light. (Stoerck.)

Bronchial breathing is very common in these locations and is not to be given undue diagnostic significance unless there are other symptoms and signs of phthisis. Individuals with thin thoracic walls are more apt to show this sort of breath sounds, while vigorous breathing and dyspnea may accentuate it. *To be of diagnostic significance, bronchial breathing must be strictly localized over a limited area and accompanied by other physical signs, especially dulness at the same spot.*

Another source of error in auscultation is the frequent changes we meet in the respiratory sounds in many patients. One day we meet at the affected area bronchial breathing, and the next day we are surprised by vesicular or feeble breathing, or complete absence of

breath sounds over the very area where distinct pathological auscultatory phenomena were audible the day before. Vigorous cough, by removing the mucous plug in some tube, may reëstablish the original sounds. I have seen such changes occurring during an examination which lasted less than half an hour. We should therefore beware of pronouncing a patient free from changes in the breath sounds before making him cough, and reëxamining the chest on several different days.

Cavernous and amphoric breathing are discussed later when speaking of pulmonary excavations and of pneumothorax.

Adventitious Sounds.—As was already stated while speaking of the technic of auscultation, adventitious sounds are to be looked for only after ascertaining the character of the breath sounds during each phase of the respiratory act. To pass judgment at the same time about both, breath sounds and rales is hazardous and we are liable to overlook many important points which are of diagnostic and prognostic significance.

The adventitious sounds audible over phthisical chests in the various stages of the disease are manifold. It can be stated that all kinds of rales—sonorous, sibilant, crepitant, subcrepitant, gurgling, etc.—are met with during the course of the disease, and each variety has some significance, indicating various pathological conditions of the lung. Paradoxical though it may seem at first sight, yet it is a fact that *there are no rales which are pathognomonic of phthisis, nor does their absence exclude the disease.* Especially is this true of the very incipience of active phthisis which, as was already intimated, begins as an infiltration and not as a catarrh of the bronchi. The neoplastic peribronchial formations may compress the alveoli; the proliferated interstitial tissues may contract and obliterate some air cells, etc., but such processes do not produce rales because at this stage the bronchi are not flooded with fluid or semifluid secretions which could interfere with the entry or exit of air through the bronchioles and air cells. Moreover, around an infiltrated area the lung usually acts vicariously, and thus veils any alteration in the breath sounds that may be created in the diseased focus, and the most we may expect is feeble, harsh, or cog-wheel breathing, but no rales.

Rales are produced when the caseous material softens and breaks through the walls of a bronchus: The secretions may irritate the bronchial mucous membrane and produce a catarrh which, in its turn, produces more secretion which, when set in motion by the passing air stream, engenders rales. This is a fact that I have had many opportunities to observe in patients who, at first, showed only alterations in the breath sounds, especially weak vesicular murmur, or cog-wheel breathing, etc., but no rales, in spite of all constitutional symptoms of phthisis which went on its course, and only later adventitious sounds made their appearance. In such cases a diagnosis of phthisis must be made without finding any rales. In fact, I have met

with acute cases in which a whole lobe was infiltrated in a comparatively short time; percussion showed distinct dulness, auscultation disclosed prolonged expiration, even bronchial breathing, but no rales at all were audible. It will therefore bear repetition that *waiting for rales, as some text-books teach, may be worse than waiting for tubercle bacilli in the sputum before making a diagnosis.*

It is worthy of mention that while rales are an indication that the tuberculous process is beyond incipiency, they do not invariably point toward an unfavorable prognosis. "Rales constitute the auscultatory evidence of inflammatory reaction to the poisons of tubercle," says Colonel Bushnell. "They are the best evidence that the lesion is resisting its foe. Rales are absent in the obsolete or arrested lesion—the body does not need to fight. They are present in the stage of reaction—the body is fighting—whether successfully or not is to be determined in part by the number and quality of the rales, in part by other considerations. They may be absent again when the body can no longer fight—when the power to react has been lost. Nothing could be more erroneous than to draw favorable conclusions from the diminution or the disappearance of rales in the very advanced case."

Crepitation.—With the onset of softening, the crepitant and, at times, the subcrepitant rale can be discovered at the affected area. The former is audible exclusively during inspiration, or only at its end, and has been compared to the sound produced by rolling one's hair between the fingers near the ear. All agree that this rale is not caused by the motion of fluid secretions in the small bronchi and air cells; nor by the explosion of air bubbles in the bronchi, as was formerly supposed. The consensus of opinion appears to be that it is caused by the inspiratory stream of air tearing apart sticky surfaces of the approximated alveolar walls, though many hold that the crepitant rale is altogether a friction sound produced by rubbing of the two pleural sheets covered with tubercles, as was first suggested by Leaming.[1] I am inclined to consider them purely atelectatic rales, analogous to those met with over the margins of healthy lungs in persons who breathe superficially, and which are often mistaken for crepitations. On the other hand, considering that apical tuberculous pleurisy is quite frequent (see page 486), these adventitious sounds are not infrequently due to frictions. The differentiation between pleural and parenchymatous lesions is discussed elsewhere.

Crepitant rales are usually audible during quiet breathing, and provoked by vigorous coughing and breathing. Moreover, they often disappear after several strong efforts at deep breathing, which would not be the case if they were friction sounds. They may be found early in the morning, and missed throughout the day, and I have seen them appear and disappear within half an hour during an examination. At times, they are heard at a very early stage of the disease

[1] Diseases of the Heart and Lungs, New York, 1884.

as quite numerous cracklings over the affected area, while in other cases but few are audible, and they are spoken of as "dry crackles," the *craquements secs* of French authors.

Crepitant rales are not by any means pathognomonic of phthisis, for reasons already stated, *but when audible over an apex showing contraction of Krönig's resonant areas, or impaired resonance in a person showing some of the important constitutional symptoms of phthisis, they are to be taken seriously.* However, in order to evaluate them properly, we must carefully study them with particular reference as to permanence during several examinations on different days and that cough does not entirely remove them. I attach greater significance to crepitant rales when heard over the supraspinous fossa, the alarm zone (see p. 379), than when heard anteriorly, above, or immediately below the clavicle, because in the latter location they are as often spurious as real. We are often able to follow them up to the stage when they become moist—subcrepitant—and finally we find that signs of excavation appear at the same spot.

During the course of phthisis, the crepitant rale is heard quite often around the seat of the main lesion, indicating that the process is extending, and over pneumonic areas so often caused by acute exacerbations. In unilateral cases, in which the other side is secondarily implicated, we may find that in the latter the first audible adventitious sounds are crepitations, and these secondary lesions are worthy of study by those who want to be able to recognize and evaluate these adventitious sounds. In fact, while teaching tuberculosis to students, advanced cases are better for this reason than early cases in which the diagnosis is often doubtful.

Moist Rales.—With the advance of the process, softening sets in and the disintegrated tubercles are eliminated from the focus through the bronchi, to be finally expectorated. These fluid and semifluid secretions, while remaining at the site of the lesion and in the bronchi, are often obstacles to the entry and exit of the air current and thus produce rales. In mild cases with but little secretion, we meet with the high-pitched subcrepitant rales produced in the small bronchi. When softening and liquefaction proceed and the secretions become more and more copious, the size of the rales increases and we hear medium, large and coarse bubbling rales and gurgles.

The difference in the size of the rales apparently depends on the difference in the size of the bronchi in which they originate—large bronchi can hold larger masses of fluid and mucous secretion, and in smaller tubes less secretions are moved, while in excavations the mass of secretion may be very large and, as a result, we get gurgles. One way to differentiate between coarse and small rales is to note their number—we commonly hear but few isolated coarse rales, while small ones are almost always in large numbers, heaps. The larger rales are more intense and louder, though of a lower pitch than the smaller, but the latter are usually more numerous, evidently because there are more

small bronchi than large ones. Rales are greater in number, and more consonating, when originating superficially, while those engendered deeply in the lung may not be heard at all. At times, we can hear rales in central lesions by placing the bell of the stethoscope in front of the patient's mouth, while all over the chest nothing is audible.

Consonating Rales.—As a rule, rales which originate in air-containing lung tissue are only faintly audible, appear distant from the ear or stethoscope, and are therefore called non-consonating. When adventitious sounds are heard very near the surface of the chest, have a more or less ringing quality, which is lacking in the non-consonating variety, they are called consonating rales; at times they possess a metallic quality, and they are designated as gurgles, or bubbling rales.

Consonating rales indicate consolidated lung tissue, a cavity or a dilated bronchus surrounded by airless tissue which either intensifies or modifies the timber of the sounds. For this reason they are at times ringing in character. On the whole it is difficult to describe them, but once heard they should not be missed in other cases. As has been shown by Sahli, better conduction through consolidated tissue is not alone sufficient to make the rale consonant; nor is loudness thus effective. Tracheal rales may be loud enough to be heard at a distance, yet they are not consonant. The consonating character apparently depends on an admixture of the higher overtones.

It may be stated that *consonating rales are engendered under the same circumstances as is bronchial breathing. They are therefore indicating that the area over which they are heard is either consolidated or excavated.* When a cavity is surrounded by consolidated lung tissue, the rales are consonating; when it is deeply seated in air-containing lung, the rales are non-consonating in character. When the cavity is large, and the secretion within them is plentiful and mobile during respiration, consolidated lung tissue around it will so intensify the sounds as to produce bubbling or gurgling rales.

Localization of Moist Rales.—It must be emphasized that no rales *per se* are pathognomonic of phthisis, because we hear more adventitious sounds in many other conditions, notably bronchitis and bronchiectasis, than in the average case of chronic phthisis. To be of significance the rales must be strictly localized over a limited area and persistent. It can be stated that, excepting in far-advanced cases, or the rare cases of chronic bronchitis complicating tuberculosis, and some forms of fibroid phthisis, *the larger the area over which moist rales are heard, especially bilaterally, the less the likelihood of their being of tuberculous origin; the higher up in the chest they are exclusively audible, the more likely that they spell phthisis; and when heard exclusively at the bases or over the lower lobes the chances that they are tuberculous are rather scanty.* Large bubbling rales, when heard over areas where there are no large bronchi, as in the upper third of the chest, are of greater significance than when heard over areas beneath which large bronchi are located. The latter may be caused by bronchitis or

bronchiectasis. When large bubbling rales are heard near the bell of the stethoscope, they are indications of phthisical excavation, because there are no large bronchi near the surface of the lung.

Sibilant and Sonorous Rales.—These are very often heard over tuberculous foci. In many incipient cases, especially in those with stationary or healing lesions, whistling and snoring rales are not uncommonly localized over one apex, especially posteriorly. When not accompanied by crackles we may take them as an indication of healing, and that they are caused by the compression of the bronchioles by fibrous tissue which forms during the process of repair. Similarly, we hear sibilant and sonorous rales over a circumscribed area as the only reminders of an old and cured tuberculous process. In senile phthisis, sibilant and sonorous rales are often the only adventitious sounds.

The asthmatic forms of phthisis, as well as those accompanied by, or implanted on, diffuse bronchitis and pulmonary emphysema, especially in fibroid phthisis, often manifest themselves by sibilant and musical rales heard during inspiration and expiration. We hear all kinds of musical notes, snoring, cooing, whistling, grunting, groaning, whining, etc. They may be heard alone while the respiratory murmur is feeble or inaudible, and then they may also be accompanied by all kinds of moist rales. Sibilant and sonorous rales or rhonchi are engendered by thick, viscid and tenacious secretions in the bronchi, or swelling or spasmodic contraction of these tubes. When heard exclusively over one of the upper lobes of a lung, they are almost pathognomonic of a tuberculous lesion. When audible all over both sides of the chest, the diagnosis of tuberculosis may not be an easy task and differentiation from chronic bronchitis, pulmonary emphysema, asthma, etc., can only be made after considering the signs revealed by percussion, as well as by the constitutional symptoms, and in some cases only the microscopic findings in the sputum can decide. When these sonorous and sibilant rales are heard unilaterally in the upper part of the chest they are easily diagnosed, as a rule.

Provoked Rales.—In many cases of early phthisis, and also at times in those with advanced disease, no adventitious sounds are heard on ordinary, or even forced, breathing; but more or less vigorous cough brings out an explosion of rales. Some writers have spoken of these as "latent rales," which is an incongruous term. Bray[1] found that in 75 per cent of cases of early phthisis, and in 30 per cent of those with moderately advanced disease, rales could be provoked by cough. *We should not pronounce a patient free from adventitious sounds unless cough has been impotent in provoking them.*

The mechanism of production of these rales has been a disputed subject. Some have suggested that they are produced by the separation of the collapsed walls of the alveoli and smaller bronchioles in and around

[1] Jour. Am. Med. Assn., 1916, **66**, 788.

the diseased focus. Bray is not satisfied with this explanation and offers the following: Toward the end of expiration the glottis is voluntarily closed and the intrapulmonary pressure is increased by powerful contraction of the expiratory muscles. This sudden increase in the intrapulmonary pressure separates the collapsed walls of the bronchioles and alveoli and the atelectatic area. Once the patency of these structures is established, the rale is produced by means of the cough, which sets into vibration the pathological secretions contained within the bronchioles and alveoli.

All kinds of rales may be provoked by cough. In early cases, some dry crackles may thus be brought out, or small, moist rales, and, at times, even showers of explosive rales may be provoked in cases in which no adventitious sounds were audible. In advanced cases large, moist, consonating rales may be brought out by cough when the bronchus leading to the cavity has been plugged, but the cough clears the passage, and permits the secretions to move with the air current. In others, sibilant or sonorous rales are thus provoked. The rales are usually heard during inspiration, but at times during both phases of the respiratory act.

Of course, no attempt should be made to provoke rales during or after a pulmonary hemorrhage, for obvious reasons.

Friction Sounds.—These are very often heard over phthisical chests. Over the apex they are heard best anteriorly above and beneath the clavicle, but here they are usually not very distinct because of the limitation of the motion of the lung in that region. Yet we sometimes perceive some grating. This is usually very difficult to differentiate from crepitation—all the criteria given in text-books are futile in some cases. At the lower parts of the thorax friction sounds are more common, especially in the axillary region. On rare occasions a pleuropericardial rub is heard not only during the respiratory phases, but also synchronous with the heart-beat. It is an indication of dry pleurisy of the lingula or other parts of the pleura in contact with the pericardium.

We distinguish friction sounds from rales by the fact that the former are heard superficially, right near the bell of the stethoscope; often they are increased by pressure of the stethoscope; they are uninfluenced by cough which usually increases the intensity of rales or entirely removes them; they are annulled when the breath is held. But the most important difference is that crepitant rales are heard during inspiration only, while friction sounds are audible during both phases of the respiratory act. However, in many cases it is quite difficult to state positively whether the adventitious sounds under consideration are of pulmonary or pleuritic origin. When found over an extensive area, especially posteriorly, or in the axillary region, frictions may be diagnosed by assuming that rales over such a large area would represent a very extensive pulmonary lesion with severe constitutional symptoms, while pleurisy may persist for years without impairing the general condition of the patient very much.

Spurious Rales.—Rales of extrapulmonary origin are occasionally heard while auscultating chests, and attributed to tuberculous changes in the lungs. In persons suffering nasal obstruction we may hear various sounds resembling rales which disappear when the patient is made to breathe through the mouth. A frequent cause of extrapulmonary rales is the falling back of the tongue when the patient makes strong efforts to breathe deeply, also after vigorous coughing the patient swallows and we believe that we hear rales in the chest.

Other spurious rales, described by Peretz[1] and William Ewart[2] in England, Colonel G. E. Bushnell[3] and Hawes[4] in this country, are caused by muscular contractions, especially the trapezius, and on raising and lowering the shoulders and arms. In persons who lift their shoulders when asked to breathe deeply these "rales" are often quite audible. French authors speak of them as *craquements et frottements sous scapulaires*, which can be heard very often over the upper part of the chest posteriorly. These muscle sounds were a potential source of error in 9.2 per cent of 250 cases examined by Hawes, while joint sounds were found in 22 per cent of cases.

J. T. King,[5] examining over 22,000 soldiers for tuberculosis in the United States Army, looked especially for these joint sounds. He kept notes of 819 men as to the incidence of spurious rales in the upper part of the chest. In 33 cases, or 4 per cent, crepitations were audible at, or near, one or more joints. Most of these sounds emanated from the scapulæ, the costosternal and sternoclavicular articulations, and from the joints at the shoulder anteriorly. In 23 instances, certain crackles, usually rather loud and explosive, were heard during one or a few respirations over the apices, disappearing promptly during continued breathing. In 17 cases, or 2.07 per cent, there were found persistent apical clicks or crackles, of the type which had often proved confusing. During the selective draft for the United States Army, several patients who consulted me after being rejected because of tuberculosis, were found to have these spurious rales in the chest. Some could not be convinced that they were not tuberculous, because many physicians told them that they have "rales" in the chest.

The so-called atelectatic and marginal rales are even more often found and must be guarded against. They are mostly heard over the anterior and lower margins of the lungs and are probably caused by the unfolding of collapsed alveoli in individuals who breathe superficially and also by the peeling off of the diaphragm from the chest wall as the lung descends into the complemental space. Richard C. Cabot[6] found them in 61 per cent of normal chests and speaks of them as crepitant and subcrepitant varieties. They usually disappear after a few breaths, but at times they persist indefinitely.

[1] British Med. Jour., 1896, **1**, 82.
[2] Ibid., 1912, **1**, 771.
[3] Medical Record, 1912, **81**, 101; **82**, 1109.
[4] Boston Med. and Surg. Jour., 1914, **170**, 153.
[5] Military Surgeon, 1918, **42**, 60.
[6] Physical Diagnosis, New York, 1909, p. 163.

CHAPTER XVII.

ROENTGENOGRAPHY IN THE DIAGNOSIS OF PHTHISIS.

Soon after the introduction of the *x*-rays, great hopes were entertained that finally a means of visualizing the condition of the thoracic viscera and detecting any changes in the lungs, bronchi, and pleura had been obtained. But after several years' experience it was found that *in tuberculosis roentgenography has its limitations*, just as other diagnostic methods. On the one hand, it does not disclose infiltrations, the very early changes in phthisis; on the other hand, because it clearly shows caseated and calcified foci, revealing airless areas of lung tissue, it helps in establishing an anatomical diagnosis. Whether the changes discovered are tuberculous in character, and whether the lesion is active, must be ascertained by other clinical methods. For this reason, roentgenography, while a very important aid in diagnosis, cannot be relied on to the exclusion of other methods. It does not disclose catarrhal conditions nor does it reveal infiltrations.

When properly used, roentgenography helps materially in disclosing certain changes in the intrathoracic viscera which formerly escaped notice during the life of the patient. Especially is this true of deep-seated lesions, pleural adhesions, enlarged bronchial glands, localized and interlobar effusions, localized pneumothorax, small cavities in the lungs, the motion of the diaphragm, abscess and gangrene of the lung, etc.

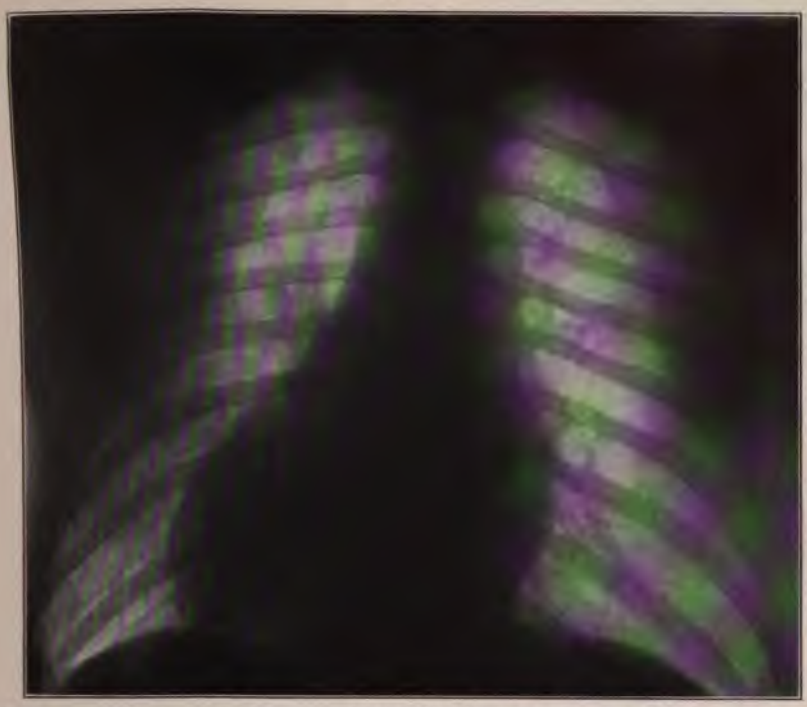
The condition of the lung and the changes at the site of the lesion in the average case of early phthisis can be made out easily by auscultation and percussion. The former even gives important indications as to the activity of the process discovered. But the *x*-rays complete the examination and often reveal deeper-lying changes in the chest which otherwise escape detection. Moreover, the practice of artificial pneumothorax, which has lately been applied with such striking success in proper cases, could not have gained general acceptance but for roentgenography.

The technic of *x*-ray examination, especially the comparative value of the various apparatus employed, will not be discussed here. This is the province of specially trained technicians. But every physician handling tuberculous cases should be able to read an *x*-ray plate and not depend entirely on the specialist roentgenographer for interpretation of the findings. *When interpreted in connection with the clinical symptoms, with which the physician alone is acquainted, the x-rays yield the best results.*

Appearance of the Normal Chest.—The appearance in the normal chest should be known before attempting to decipher pathological

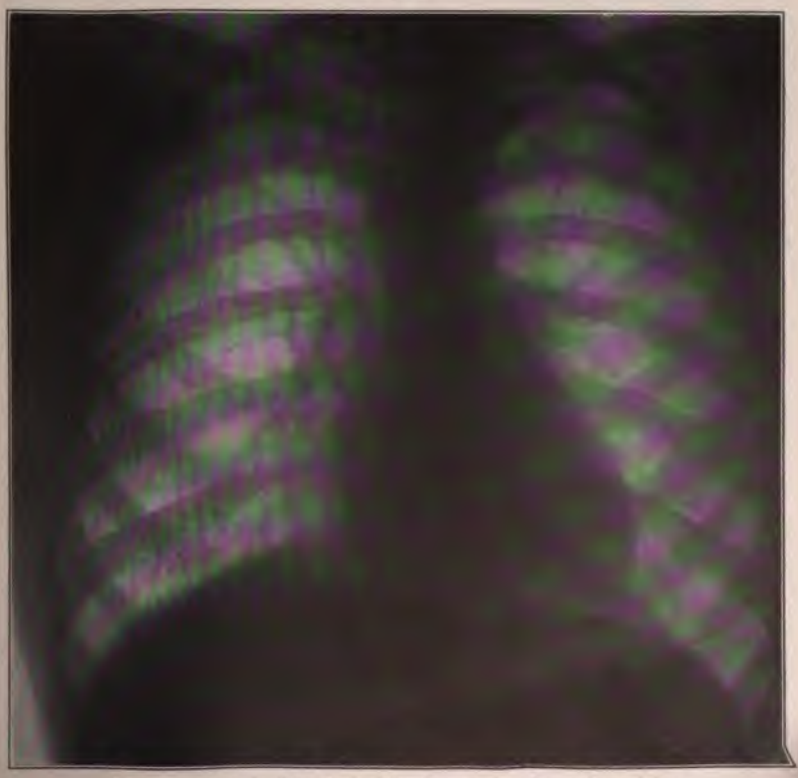
PLATE X

FIG. 1



Roentgenogram of a man with apparently healthy thoracic viscera. Dorsoventral position.

FIG. 2



Same man as in Fig. 1, but in the ventrodorsal position.

changes. It is, however, a fact that a normal chest, showing no signs suggestive of pathological conditions, is exceedingly rare. I have not yet seen one. Plate X shows plates from a chest of a man apparently free from pulmonary disease.

While passing through the thorax, the rays are obstructed by the various tissues, according to their density, volume and constituent elements, and the result is that the denser tissues cast shadows on the screen or plate. The ribs cross the lung fields appearing as dark, sharply defined bands, their posterior parts are seen forming a concavity downward, while the anterior parts are convex downward. In normal healthy young persons the costal cartilages are not all visible.

FIG. 81.—Structures making up the hilus shadow: *R*, second rib; *W*, second thoracic vertebra; *V*, arch of azygos vein; *B*, bronchus; *L*, bronchial lymphatic glands; *A*, aorta; *P*, pulmonary artery; *O*, esophagus; *D*, thoracic duct. (Doyen.)

However, early ossification of these cartilages is consistent with healthy lungs in many cases. The clavicles produce a distinct shadow, running horizontally outward and slightly downward. In some persons the clavicles overlap the first ribs and thus obscure the lung apex. The scapulæ also are visible as more or less distinct triangular shadows.

The densest shadow seen is that of the heart and great vessels in the middle and to the left, and the diaphragm beneath. Because it permits the rays to pass with less resistance than any other organ in the chest, the lung gives a dark image on the negative; the heart, the large vessels, the diaphragm and the liver, because of their density

and blood content, obstruct the rays and produce light areas on the plate. The most translucent parts of the healthy viscera are the healthy lungs; when they are collapsed by air in the pleura, as in pneumothorax, the space is even brighter. In healthy persons, when the patient takes a deep inspiration, the lungs brighten up. But the brightness of the lung tissue is not absolute. There is seen a delicate, at times even a more or less coarse, arborization, as of a network, passing from the roots of the lung to the periphery. At the roots it is caused by the greater density of the tissues, but in most persons also by the deposition of carbon particles, which may be found in nearly every individual over fifteen years of age. When the shadow at that point is abnormally accentuated, it may be an indication of enlargement or calcification of the glands, and in children it points to tuberculous tracheobronchial adenopathy. Often we note in this region small, sharply defined, oval opacities which represent optical sections of bloodvessels.

It is, however, difficult or impossible to evaluate every shadow or opacity because by their passage through the chest the rays are obstructed by the various parts constituting the viscera, thus producing superimposed shadows. Carefully prepared stereoscopic pictures may enable us to distinguish these superimposed shadows in perspective, but they are after all not much superior to a good roentgenogram taken by instantaneous exposure.

The Hilus Shadow.—The shadows seen at both sides of the heart are very frequently a source of confusion in diagnosis. As will be seen from Fig. 81, they are due to the density of the tissues composing the bronchi and the large vessels, which are seen either in transverse, or in optical section, combined with the opacities produced by the regional lymphatic glands and connective tissue, none of which can be differentiated on the screen or plate. While in some cases circumscribed opacities, or spots, represent calcified glands or nodules, in others they are produced by deposits of dust in the peribronchial lymphatic tissues which are very frequent in adults, and even in children in cities they are not uncommon. But in many cases simple engorgement of these tissues with blood is apt to give a shadow in that region. In fact, during attacks of measles or whooping-cough the glands in the chest have been found visible in roentgenographic plates, and the same is often the case in acute affections of the respiratory tract in children or adults.

It is thus clear that many conditions other than tuberculosis of the tracheobronchial glands may cause shadows or opacities in the hilus region. Moreover, even when these opacities represent anthracotic or calcareous glands, the roentgenogram alone gives us no clue as to the activity of the process, which is after all the main problem in clinical diagnosis. In children it is hazardous to diagnosticate tracheobronchial adenopathy because of these opacities when the clinical picture is not in agreement.

To the right side of the heart the hilus shadow is more extensive than to the left because in the latter location the heart shadow obscures the hilus structures. In many cases we see strands passing from the hilus to the periphery, or the diaphragm. It is the consensus of opinion that they are produced by bloodvessels, and occasionally by bronchi which at times appear in optical section.

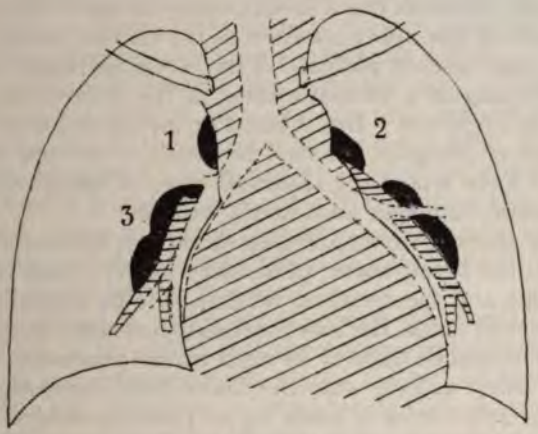


FIG. 82.—Diagrammatic representation of the location of the thoracic glands based on numerous observations of roentgenograms compared with anatomical findings. Only those at the roots of the lungs are represented in black, while those that are covered by the pulmonary artery and the roots of the bronchi and mediastinum are left out. 1, paratracheal gland; 2, tracheobronchial gland at the aorta; 3, bronchopulmonary lymphatic glands at the hilum on both sides. (Assmann.)

Fluoroscopy.—In the vast majority of cases of tuberculosis, and in suspects, a fluoroscopic examination is sufficient for diagnostic purposes; in the few cases in which a plate is desirable, fluoroscopy is not to be neglected, because it gives us information which the plate does not. Fluoroscopy is to be preferred in many cases because it shows the motion of the parietes of the chest, of the diaphragm, and of the pulmonary apices, etc. The room in which fluoroscopy is done must be totally dark, and inasmuch as this is very difficult to attain in the average physician's office, it is best done in the evening.

The following points are to be looked for on the fluoroscopic screen when examining a chest: The ribs; the median shadow; the diaphragm; the hilus shadow; the space above the clavicle are to be carefully studied.

In healthy and well-formed individuals the ribs are seen symmetrically placed on both sides, moving with each respiratory act. Unilateral limitation of motion of the ribs is suggestive of unilateral disease, and phthisis is to be thought of in this connection. When we find the ribs on both sides unduly horizontal we should look for pulmonary emphysema; when the horizontal setting is unilateral, while

the lung markings are absent, pneumothorax is to be suspected. Normally, especially in young subjects, the costal cartilages are not distinctly visible on the screen. The ribs are sharply cut off (see Fig. 2, Plate X). In older persons they are usually visible, owing to ossification which takes place with advancing age. In tuberculous patients ossification of the costal cartilages, especially the first (Fig. 1, Plate XVI), is very frequently seen on the roentgenogram. As was already stated, Freund considers this a predisposing factor to phthisis because of the stenosis of the upper aperture of the thorax, which it is apt to cause. In some cases of phthisis all the costal cartilages are calcified, and when looking at a patient's chest in the fluoroscope, this point should not be neglected. But it must be mentioned that it is not an infallible sign of phthisis. It may be found in persons who are not sick, while I have repeatedly observed cases of advanced phthisis in which the costal cartilages were hardly visible.

Within the thoracic cavity the deep shadow representing the mediastinal organs, the heart, aorta, pulmonary artery, vena cava, as well as the sternum and the vertebral column, is to be carefully examined. It is triangular in shape, the base extending markedly to the left of the sternum. The middle third of its right border represents the superior vena cava: when bulging out, the lower third represents the right auricle. The left border is made up of three successive convexities: The first is produced by the arch of the aorta; the middle, the pulmonary artery and the left auricular appendix; while the lower is the left ventricle of the heart. All or any of these convexities are seen, in many cases, to throb rhythmically; at times the succession of the beats of the ventricle and those of the pulmonary artery may be seen very clearly. In phthisis the heart is, as a rule, smaller than normal.

The hilus shadow, on both sides of the median shadow, should be carefully studied. It is best seen in the right side because in the left it is in part covered by the heart. As was already stated, its significance is very frequently overestimated by roentgenologists. In healthy individuals it represents primarily the bloodvessels, the bronchi, the bronchial glands, and connective tissue which is abundant in that region. It is the consensus of opinion that this shadow merely indicates the absence of air and gives no clue as to each individual structure which enter into its make up. When the thoracic glands are enlarged or calcified, the hilus shadow appears larger and more accentuated. This point is discussed elsewhere in detail.

The lungs are seen within the thoracic cage as two triangular bright fields; the upper part is separated from the rest by the shadow of the clavicle above which the lung apex can be inspected. The base is delimited by the diaphragm, which moves with each respiratory act, being raised during expiration and lowered during inspiration.

The apices are carefully inspected, and the translucency of the lungs in these regions inquired into. Theoretically, it should be of equal intensity on both sides, but such perfection is only rarely

encountered, even in healthy persons. Usually, owing to thickness of the muscles, scoliosis, etc., one side is somewhat darker. But this is best studied on the roentgenographic plate. With the fluoroscope we look for the "cough phenomenon," first described by Kreuzfuchs.[1] This author noted that in healthy individuals the translucency of the apices varies according to various conditions, especially the form of the chest. Deep respiratory efforts may clear up any shadow in healthy lungs. During cough the apices brighten up even when they are otherwise quite dark, excepting when there is diseased tissue in that region and the affected apex remains dark even during cough.

But this is not a very reliable sign. Jordan[2] says: "Failure of the apex to light up is difficult to make out with certainty; there are endless fallacies due to the position of the *x*-ray tube, the thickness of the pectoral muscles of the patient, the 'lie' of the ribs and clavicle, etc., and at best it is almost impossible to reproduce this 'failure' on a photographic plate with any certainty. I am quite sure that we should diagnose pulmonary tuberculosis in a large number of healthy subjects if we are to rely on this sign."

This view is shared by many, but it appears that Jordan is mistaken in his statement to the effect that the cough phenomenon cannot be reproduced on a roentgenographic plate. As will be noted on Plate XII, F. Holst[3] has succeeded in reproducing this phenomenon very clearly. Moreover, this author has also shown that during cough there is an alteration in the lateral limits of the pulmonary apices, they become wider while the trachea becomes narrower, sometimes as much as 1 cm. In normal individuals this phenomenon is observed on both sides to the same degree, while in case one apex is altered by tuberculous changes, it fails to brighten up, and remains narrow and darker during cough. Of course, this phenomenon is best studied on the screen, and only exceptionally may it be reproduced on a roentgenographic plate. We must, however, guard against mistaking the apparent changes in the brightness of the apices during cough caused by the separation of the ribs and widening of the intercostal spaces. It has been of immense service to me in many cases.

With the aid of fluoroscopy we also ascertain the size and position of the heart. In phthisis this organ is, as a rule, smaller than normal. In fact, when I find a large heart in a dubious case I hesitate before making a diagnosis of phthisis. In phthisis it is also very often vertical; it may be "hanging," cardioptosis, and in more advanced cases frequently displaced toward the affected side.

After the apices, the diaphragm should claim our attention. The mobility of this muscle has been found defective on the affected side in many cases of phthisis; according to F. H. Williams,[4] in the very

[1] München. med. Wchnschr., 1912, **59**, 80.
[2] Lancet, 1914, **1**, 963.
[3] München. med. Wchnschr., 1912, **59**, 1659.
[4] Am. Jour. Med. Sci., 1897, **114**, 655.

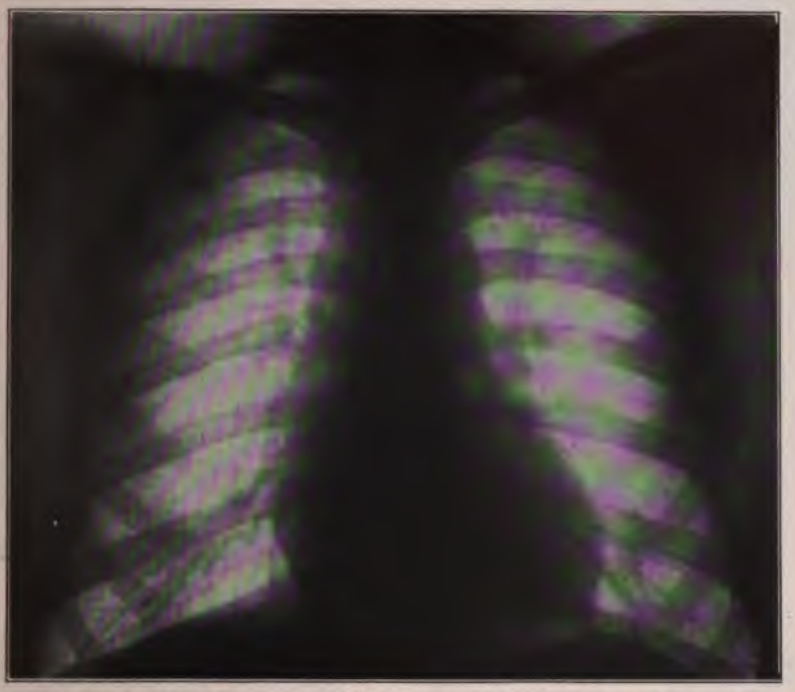
incipient stage. The motion of one-half of the diaphragm may not only be delayed when there is a pulmonary lesion, but it is at times seen to be "jerky," or "stammering," as Harold Mowat says. In some healthy persons the mobility of the diaphragm is very limited, while in most the breathing excursion is from three-fourths to one inch, and during forced respiration it may even move more than two inches, the left half of the muscle more than the right. When both sides are stationary it may indicate emphysema, or nothing at all, but when one side moves while the other is immobile or its excursion is relatively limited, we should suspect tuberculosis. Various explanations have been given for this phenomenon. Some have attributed it to diminished power of retraction of the lung, others to implication of the terminal branches of the vagus, or of the phrenic nerve in apical pleural thickenings, etc. In advanced cases limitation of motion may be due to pleural adhesions. It must, however, be emphasized that in itself defective movement of the diaphragm may be found in healthy individuals. If unilateral it may be due to paresis of that muscle, or to an old basal pleurisy producing adhesions which hinder its excursion. In persons with big abdomens, the breathing is usually purely thoracic, and the diaphragm is immobile.

Extensive experience has shown limitation of motion on the affected side of the diaphragm in only a few cases of incipient phthisis. Indeed, we often see advanced cases in which both sides of the diaphragm are freely and equally mobile. On the other hand, limitation is found in non-tuberculous cases owing to adhesions remaining after previous attacks of pleurisy the individual being quite healthy at the time. In advanced cases this phenomenon has to be considered in connection with the feasibility of artificial pneumothorax, but, as will be shown later on, it is not absolutely reliable.

At the outer extremities of the diaphragm are the *costodiaphragmatic sinuses*. They should be examined carefully in every case, and both sides should be compared. The lower angle of the sinus should be long and sharp; during inspiration it enlarges and brightens up; it contracts and loses its brilliancy to some degree during expiration. Any diminution in its size, or obtuseness of its apex, or its complete obliteration, indicates a pathological process of the pleura or lung. The two sides should then be compared, but it must be borne in mind that in the right side the liver makes it somewhat smaller, while in the left side the air bubble of the stomach may alter it to some degree. The angle formed by the heart and the liver, the *cardiohepatic angle*, often appears obtuse, or obliterated in tuberculosis, especially pleurisy, or thickened pleura. The dome of the diaphragm is also changed by a thickened pleura; it is no more smooth, but shows marked elevation of the curve during inspiration; in others, we note a series of small irregularities in the contour; in still others, bands of connective tissue are seen passing from the diaphragm to the lung.

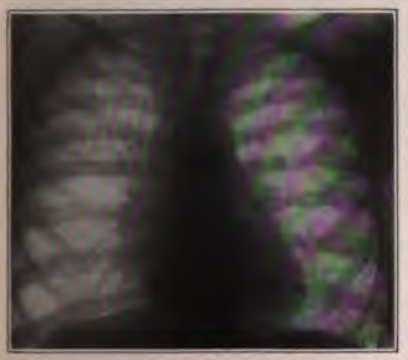
PLATE XI

FIG. 1



Roentgenogram of a woman with apparently healthy thoracic viscera.

FIG. 2



Roentgenogram of the chest of a child eight years old. Though no symptoms or signs of tracheobronchial adenopathy could be found clinically, the roentgenogram shows shadows suggestive of such a condition.

FIG. 3

Roentgenogram of a child nine years old, suggestive of enlarged hilus glands. The symptoms and signs of this disease were, however, lacking. On a level with the second rib an opacity suggestive of a calcified gland can be seen.

PLATE XII

FIG. 1

Lung apex during ordinary breathing.

FIG. 3

Apex during ordinary breathing.

FIG. 2

The same apex while patient is coughing, and showing a narrowing of the trachea, widening, and lightening up of the apices, especially the right. (F. Holst.)

FIG. 4

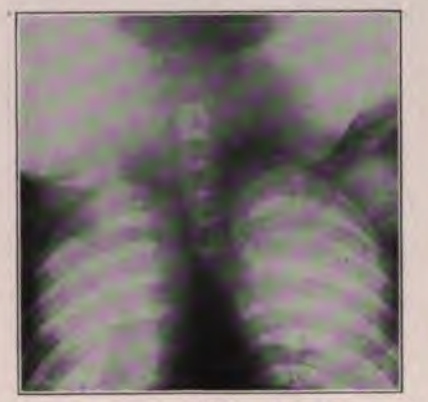
The same apex while patient is coughing, showing narrowing of the trachea and lightening up of the area of the lung. (F. Holst.)

PLATE XIII

FIG. 1

Roentgenogram of a case of abortive tuberculosis. Though suggestive of an extensive lesion in the left apex, the physical signs, as well as the course of the disease, showed that the activity of the process was benign. The patient recovered within three months.

FIG. 2



Roentgenogram of the apices in acase of incipient phthisis. No definite changes are visible, though physical exploration revealed a distinct lesion in the left apex, and the constitutional symptoms were clearly those of phthisis.

FIG. 3

Slight infiltration of the right apex. Marked increase in lymphatic tissue in both hilus regions.

FIG. 4

Partial consolidation of both apices, large cavity in left apex and thickened interlobar fissure. Dilatation of bronchi of lower lobe of left lung. Heart displaced to the left.

PLATE XIV

FIG. 1

Fibroid changes in right apex, which some call an "incipient lesion." Hilus shadow strongly marked. Peribronchial fibrosis.

FIG. 2

Fibrocaseous lesion in right apex. Peribronchial fibrosis visible at each hilus.

FIG. 3

Tuberculous lesion of the fibroid type in the right upper lobe. Sputum positive. Substernal thyroid protruding into the upper third of the left chest.

FIG. 4

Miliary tuberculosis of the upper lobe of the right lung. Substernal thyroid in the left side.

PLATE XV

FIG. 1

Fibrocaseous lesion in right apex. Peribronchial infiltrations and calcified glands at the hilus on both sides.

FIG. 2

Very dense infiltration of right upper lobe and large cavity below the clavicle limited below by the thickened interlobar fissure. Marked peribronchial infiltrations. The hilus region on both sides shows old fibroid and calcified glands.

FIG. 3

Large cavity surrounded by a dense fibrous wall in upper part of right lung. Enlarged glands in right hilus region. Lower half emphysematous. Left lung shows moderate infiltration beneath the clavicle and enlarged hilus glands. Drop heart.

FIG. 4

Bilateral tuberculous infiltration of both lungs. Dense hilus region due to calcification of glands. Several small cavities in right lung. Adhesions of diaphragm. Trachea markedly pulled over to the right. Stomach visible at left base.

PLATE XVI

FIG. 1

Moderate caseation of both apices. Coarse infiltration of lower half of left lung with thickened pleura. Heart pulled over to the left and downward. Emphysema of right lung. Diaphragm in right side shows a bulging due to adhesions.

FIG. 2

Diffuse caseation of upper third of left lung. The rest presents a dense homogeneous shadow caused by consolidation of pulmonary parenchyma as well as thickened pleura. Right lung emphysematous and several calcified glands are seen at the hilus.

FIG. 3

Chronic cavitary phthisis in a child eight years of age, with displacement of the heart to the left.

FIG. 4

Anthracosis and fibroid phthisis. Diffuse nodular infiltration of both lungs with multiple cavitation. "Honeycomb" appearance.

PLATE XVII

FIG. 1

Cavity in upper lobe of right lung filled with secretions.

FIG. 2

Same patient as in Fig. 1. Secretions emptied from the cavity by expectoration.

FIG. 3

Large cavity in upper lobe of the right lung with fluid level. Partial pneumothorax which has not been effective in compressing the cavity.

FIG. 4

Partial pneumothorax of upper half of left lung with fluid level. Emphysema of right lung.

PLATE XVIII

FIG. 1

Dense consolidation of lower half of right lung with thickened pleura. Large cavity in left lung occupying apex on a level with first two interspaces. Drop heart.

FIG. 2

Diffuse caseation of both lung apices. Round cavity, surrounded by a dense fibrous capsule, under the right third interspace in mammillary line. Irregularity of the diaphragm due to adhesions.

FIG. 3

Large, oval-shaped cavity in right upper lobe. Lymphatic tissue at hilus increased. "Annular shadow" in middle portion of left lung at third interspace. Heart dropped; pleuropericardial adhesions.

FIG. 4

Vertical heart. Multiple cavitation in right upper lobe, "honeycombed." Lower part emphysematous. Small cavities in left upper lobe. Marked hilus changes.

Roentgenography.—Of great value in all stages of phthisis, especially in dubious early cases, and in those in which a permanent record is desired, is roentgenography. When properly taken and developed, the plate may be studied at leisure and slight alterations, which are not visible on the fluoroscopic screen, may be detected.

In evaluating the roentgenographic findings we must bear in mind the following points: Small infiltrations do not show any definite and clear-cut signs on the plate; at any rate, the shadow they cast is not pathognomonic. Cohn[1] inserted tuberculous tissue into healthy lungs of cadavers, of which he took roentgenograms and found that 1 cc of diseased tissue is not visible on the plate. Ziegler and Krause[2] have investigated the problem and found that pieces of tissue less bulky than 4 cc are not visible on the roentgenogram, and that, on the whole, small areas of infiltration are only visible when they are located near the surface of the lung. Experimental investigations of Joseph Walsh, James W. Wood and J. C. Thompson confirmed the above findings.[3] In other words, *small infiltrations, when centrally located, are screened by normal pulmonary tissue, and may escape detection. When the lesion has caseated it casts a more or less dense shadow.* But then the case is no more incipient.

In many cases we find that the affected apex is darker than its mate on the opposite side. In others, the affected area has the appearance of "ground glass." But even this does not invariably imply an active lesion. Indeed, it may be put down as a general rule that, in suspicious cases showing no constitutional symptoms, the darker the apex, the less likely the probability of its being a sign of *active incipient tuberculosis*. It may be revealing an old and healed lesion. Walsh and his coworkers, comparing lungs removed, with roentgenographic plates taken during life found that the greater the amount of fibrous tissue, the greater the density of the shadow. I have been impressed with the following fact: A considerable proportion of apparently healthy people have one apex, usually the right, darker, due to various causes. In many it represents healed tuberculous lesions which are no longer active, nor serious. When in these individuals there occurs a new tuberculous lesion in the opposite apex, which is not uncommon, it is responsible for the constitutional symptoms calling for a roentgenographic examination. The report from the roentgenographer may state that the lesion is located in the right side, while the physical signs show conclusively that the active lesion is in the left, or the reverse.

The divergence of findings on physical examination and roentgenography is best seen in far-advanced cases of phthisis in which a new lesion occurs in the hitherto unaffected apex. The plate does not show it distinctly until caseation has taken place, while physical exploration reveals it clearly. I have had this incontrovertible proof of the inade-

[1] Ztschr. f. Tuberkulose, 1911, **17**, 217.
[2] Röentgenatlas der Lungentuberkulose, Würzburg, 1910.
[3] Tr. Nat. Tuberc. Assn., 1919, **15**, 69.

quacy of roentgenography in incipient lesions repeatedly. Joseph Walsh also found that "physical signs have frequently brought out lesions and extensions invisible on the *x*-ray, while the latter have shown only rare ones not found by the former."

For these reasons we should not conclude merely on finding opacities in one apex that we are dealing with a case of active incipient phthisis. When found in connection with constitutional symptoms and signs on physical exploration these opacities are of diagnostic value. Nor should we conclude in the presence of constitutional symptoms and local signs suggestive of phthisis, but negative roentgenographic findings, that a case is not tuberculous. Such a case requires further observation, despite the negative *x*-ray findings. I do not hesitate to make a diagnosis of pulmonary tuberculosis under such circumstances when clinical evidence warrants it.

After the apex we carefully examine the condition of the roots of the lungs, the hilus, with a view of ascertaining the presence of enlarged caseated or calcified glands or peribronchial infiltrations in that region. The shadows and mottlings observable at these points have been discussed. At first there was a tendency to consider all abnormalities as evidences of enlarged glands, and a diagnosis of tuberculosis, or tuberculous adenopathy, was made on this evidence alone. But experience has shown conclusively that this shadow may be caused by any congestive condition and especially by fibrosis due to various causes of the glands, bronchi and lungs, and it is not pathognomonic of phthisis. There is hardly an adult living in a city, or working at a dusty trade, who has no peribronchial thickening, enlarged, or calcified, glands at the hilus of the lungs. It was also found by Cohn, Dunham, Boardman, Wolman, Bibb and Gilliland, and others, that except in cases with calcified glands, these shadows are caused by blood in the vessels of the thorax. Blood is known to absorb the *x*-rays more readily than infiltrated soft tissue or sputum. Experimental injection of the arteries in the lung has been found to intensify the shadow, and in human beings injection of the vessels with substances giving a strong shadow produces pictures which are exactly like those of normal lung markings. But even this interpretation has recently been challenged by Joseph Walsh and coworkers. He found experimentally that an artery the size and thickness of the carotid casts so slight a shadow even when filled with blood that it is likely the blood-vessels *per se* have little or nothing to do with the hilus shadow, or normal lung detail. It is his opinion that the hilus shows up, not on account of specific structures, but merely on account of lack of air content.

These facts explain many of the thickenings and strands noted on chest plates, running from the hilus to the periphery of the lungs. In some cases they are due to bronchitis with congestion; in others, the mottling is due to strands of connective tissue, or calcified glands which though tuberculous in origin, still are harmless and of no clinical

importance. Sewall and Childs report the case of a presumably non-tuberculous stone-cutter furnishing a roentgenogram in which, except for the relatively moderate involvement of the apices, the mineral deposits occasioned opacities resembling the densest tuberculous structure. I have often had the same experience with workers at dusty trades. The criterion given by some authors for distinguishing inactive consolidations and calcified glands from shadows representing active lesions by the fact that the latter appear "wooly," does not hold in many cases. Any structure out of focus appears diffuse—"wooly"; even instantaneously taken plates are not free from this source of error. In most cases when we find mottling, especially surrounding extensive lesions we are justified in considering them tubercles and an extension of the process; but we have seen many cases in which the plate showed extensively scattered tubercles, "a thick snowstorm," yet at the autopsy no miliary tuberculosis was found. Miliary carcinomatosis of the lung gives a similar picture on the plate. I have had several patients with plates taken by radiographers which showed these scattered tubercles, thus inducing these *x*-ray specialists to make diagnoses of miliary tuberculosis. Yet these patients walked into my office for examination, were found afebrile, and the subsequent course indicated that they had no real miliary tuberculosis. "The interpretation of less dense and more diffuse opacities is chiefly guesswork," say Sewall and Childs.[1] "They usually represent either pathological lymph nodes or bloodvessels in more or less optical section."

Sources of Error.—The analysis of these shadows and mottlings admits of so many interpretations that they are of doubtful utility in most incipient cases. The "ground-glass" appearance of an apex is found in plates taken from healthy individuals and no symptoms indicating caseation of lung tissue can be found on prolonged clinical observation. A shadow in the apical region, when not the result of scoliosis, shows that there is some airless tissue in that location. But we are not justified in invariably assuming that it was caused by a tuberculous infiltration; or even if so, that the lesion is active. Ziegler and Krause, Dehn, Arnsperger, Assmann, and others have found that calcified and caseated tissue, and even fluid, anthracotic and calcified lymph glands, and most commonly connective tissue produce the same roentgenographic shadows. I have seen a large empyema failing to disclose itself on an *x*-ray plate.

There is no more justification for placing an individual, one of whose apices casts a shadow on a plate, under prolonged and costly treatment than there is for the treatment of one for mitral insufficiency merely because a systolic murmur is audible at the cardiac apex. In both cases the clinical symptoms decide whether the person is sick and in need of treatment.

[1] Arch. Int. Med., 1912, **10**, 45.

Because we are looking, in incipient cases, for small areas of recent infiltration, it is clear that we cannot rely on roentgenography alone for the diagnosis of early phthisis. The roentgenographic picture gives the history of the thoracic viscera throughout the life of their owner. Any pathological change which may have occurred at any time may have left traces behind which are likely to cast shadows or cause opacities on the plate. For this reason, in incipient or dubious cases, the roentgenographic findings are to be taken only in connection with constitutional symptoms and physical exploration of the chest. If the latter are negative, the case is to be considered non-tuberculous, no matter what the roentgenographic plate shows.

It is thus clear that in the diagnosis of incipient phthisis the *x*-rays are not of the value which some authors have attributed to them. Early tuberculous lesions, slightly enlarged bronchial glands, unless caseated or calcified, as well as mucous secretions, usually permit the rays to pass through without casting any shadows on the plate. Optical sections of bloodvessels, due to any condition that may cause vascular engorgement, may show opacities on the plate simulating the characteristics of tuberculous lesions and may lead to error.

What is of most importance in obscure lesions is not so much their causation but their activity. A healed tuberculous lesion in an apex is not incompatible with excellent health, as has been repeatedly emphasized. But the cicatrix produced by the healing process produces a shadow on the roentgenogram as well as, and often more striking than, an active lesion.

Roentgenography may be of great assistance in attempts at localization of a lesion, though smaller tuberculous foci may often be discovered with the orthodox clinical methods of diagnosis, and the determination of the activity of an apical process can only be accomplished by careful observation of the case, paying special attention to the constitutional symptoms, such as the temperature, the pulse, cough, expectoration, and the physical signs. "With our present ability to produce and interpret *x*-ray pictures," say Sewall and Childs, "it must be admitted that a judgment founded on clinical history combined with physical signs may lead to a strong suspicion of tuberculous infection long before any signs of actual tissue changes, except those involving bronchial glands, appear on the *x*-ray negative." Wolman,[1] who has worked with the stereograph, arrives at a similar conclusion. He says: "In the great bulk of cases the stereograph tells us no more than a careful clinical examination, yet in a fair number of cases, and those among the most interesting and puzzling, it gives additional information. But we must add the caution that a careful history is indispensable, since not even the stereograph can tell an active from a healed lesion." Likewise Joseph Walsh says, "Advanced tuberculosis with massive fibrosis or caseation, numerous tubercles, or large cavitation can

[1] Johns Hopkins Hosp. Bull., 1911, **22**, 236.

usually be diagnosed by the *x*-ray, though still more accurately by physical signs. The best results are achieved by careful physical diagnosis compared with the *x*-ray plate and, if differences are found, submission of these to physical examination before conclusion is reached. Physical signs have frequently brought out lesions and extensions invisible on the *x*-ray; while the latter have shown only rarely ones not found by the former."

When my findings on physical exploration of the chest disagree with the roentgenographic shadows and interpretation, I place more reliance on the purely clinical methods, and quite extensive experience, with many autopsies have taught me that careful and *competent* physical diagnosis is more reliable in doubtful cases than roentgenography.

Roentgenography in Advanced Stages of Phthisis.—In my experience roentgenography has been of greater utility in the diagnosis of advanced disease than in early or dubious cases. However, only rarely we find that the *x*-ray plate reveals more extensive involvement than the findings on physical exploration of the chest. In cases in which the question of artificial pneumothorax is considered, roentgenography offers invaluable assistance. Very often pleural effusions, especially the localized or interlobar varieties; pneumothorax and pleural adhesions, are discovered, though they have escaped detection by routine methods. The same is particularly true of localized pneumothorax.

The roentgenographic picture of advanced phthisis is variegated, depending on the changes in the lungs and pleura. The intensity of the shadows cast by the lesions depends on their nature and density. Caseated and calcified areas cast dense shadows, while proliferation of tissue, especially when it is also congested, or fibrosis, is also clearly detected. Old, indurated areas are usually more or less sharply demarcated from the surrounding tissues, while with new, active infiltrations the shadow merges by degrees with the surrounding air-containing lung tissue. Thick pleura is discovered by a dense, uniform shadow, and all connective-tissue formations reveal themselves in the same manner. More often than by physical exploration, cavities disclose themselves by showing limited areas lacking in lung markings and surrounded by thick shadows (Plates XV, XVII). They may often be seen moving during inspiration and expiration when examined with the fluoroscopic screen. But when a cavity is filled with secretions it is again airless and casts the shadow of the surrounding tissues, and a very much thickened pleura may cover up a cavity. The difference in appearance of a cavity when filled with secretions, and then when the secretions have been brought out by expectoration is well shown on Plate XVII. A cavity may also be screened by the surrounding healthy lung tissue. Thus, we often fail to find it with the *x*-rays, while physical exploration reveals it easily. Sampson, Heise, and Brown[1] have recently shown that in many cases the annular or ring-

[1] American Review of Tuberculosis, 1919, **2**, 664.

like shadows seen often in almost normal or mildly infiltrated lung fields are no indications of intrapulmonary cavities, as has been supposed by many, but altogether localized, usually interlobar pneumothorax. The writer has not been able to confirm these findings. In several cases these annular shadows were clearly marked on the roentgenograms, yet neither cavities nor interlobar pneumothorax were found.

The differentiation between thick pleura and pleural effusions is very difficult in most cases; in many impossible, if we rely on roentgenography alone. The following rule may be of assistance in some cases: When the signs found by percussion show a more extensive lesion than the roentgenogram shows, then it is the thickened pleura which produces the dulness. Conversely, when the signs obtained by percussion indicate a lesion of slighter extent than the roentgenogram reveals, there is a central parenchymatous lesion of very serious import.

The condition of the pleura may be studied on the plate. Fibrinous pleurisy is not shown at all, and very often quite a thick pleura, easily discovered by physical diagnosis is not seen on the plate. I have seen at an autopsy quite a thick pleura, about 1 cm. in thickness, which gave no indication of its presence on the roentgenographic plate. But an effusion reveals itself clearly as an intense homogeneous shadow on the plate. Its upper level is not clearly demarcated from the lung above, and in the fluoroscope it may be seen moving somewhat with the respiratory movements. When the quantity of fluid is small, it may escape detection when sinking down in the diaphragm. In hydropneumothorax it is important that the exposure should be made with the patient in the erect posture, because when lying down small quantities of fluid spread in a thin layer and may escape detection. In hydropneumothorax the upper layer of the fluid forms a sharp line, while in pleurisy with effusion the upper level is usually not so sharp, but gradually merges with the lung tissue above it. The fact that in the latter case the level does not shift with motion of the patient's chest shows that it is not a hydropneumothorax; in the latter case it does shift (see Plate XXIII).

The displacements of the mediastinum caused by pleural effusion are best made out with the *x*-rays; but it is impossible to distinguish between fluid and the liver in right-sided effusions. Dislocation of the trachea and larynx may often be discovered on the plate (Plate XV).

CHAPTER XVIII.

THE CLINICAL FORMS OF PHTHISIS.

POLYMORPHISM OF THE CLINICAL PHENOMENA OF PHTHISIS.

LAENNEC showed clearly the unity of the elemental pathological changes occurring in phthisis, and Koch, discovering the tubercle bacillus, proved it etiologically. But all attempts to impose this unity on the clinical manifestations of tuberculous diseases of the lungs have failed dismally. In pathology, particularly in clinical medicine, unity of causation does not always indicate unity of effect. Especially is this true of a polymorphous disease, as pulmonary phthisis.

A study of the morbid anatomy of phthisis shows great polymorphism—there are hardly two cases showing the same changes in structure. There are cases in which the lesions are purely proliferative, characterized by the formation of tubercles, as is the case with acute miliary tuberculosis; in others they are mainly exudative, as in chronic phthisis. But in the latter the difference in the intensity of the productive inflammation, which tends to limit the morbid process; and the process of necrosis, which tends to extend it, produce a diversity of lesions which have important bearings on the clinical picture, course, and prognosis of the disease.

This is to be expected when we consider that the disease produced by the tubercle bacilli depends on the interaction of two forces of inconstant intensity, viz.:

1. *On the Intensity of the Infection.*—This depends on the number of bacilli which have entered the body; their virulence which we know is variable, depending on the type and the condition under which they existed before entering the body, etc., and on the portals of entry. It is doubtful whether infection by inhalation will produce the same clinical picture as infection by ingestion or by inoculation; whether hematogenous tuberculosis will produce the same symptoms as aërogenous or lymphogenous infection.

2. *On the resistance of the host,* which is also an inconstant value, depending as it does on so many variable, complex, certain and uncertain, constant and transient conditions which cannot always be defined clearly. Thus, the effects of the infection depend on the dose, and also on the age at which it has taken place. During the first six or twelve months of life massive infection produces a different disease from that of the succeeding years of childhood. Acute miliary tuberculosis is common at the former age, while tuberculosis of the glands, bones, and joints is mostly seen at the later ages. Primary infection

of an adult is usually followed by clinical phenomena which differ markedly from those seen in individuals who were presumably infected during childhood, and the bacilli remained dormant for many years. We have already discussed the effects of preëxisting diseases on the type and course of phthisis.

"To speak of pulmonary tuberculosis as an entity," says von Hansemann,[1] "and to describe it as one disease, caused by the tubercle bacillus is hardly conceivable. One has to compare pure miliary tuberculosis of the lungs with chronic indurative phthisis, and the latter with acute florid phthisis, or caseous hepatization of the lungs, to find clearly that they are different pathological pictures which defy all comparisons. For these reasons it is altogether impossible to speak simply of pulmonary tuberculosis and thereby retain a clear survey of the different forms of the disease. In reality we are compelled to draw a sharp line of demarcation between these different forms of the disease, even when we are inclined to consider the tubercle bacilli as the underlying etiological cause of all the forms of the disease."

The Stages of Phthisis.—Early writers on phthisis, who were innocent of modern methods of diagnosis, felt constrained to differentiate various forms of the disease as they saw it clinically. They divided it into three stages: *Phthisis incipiens, phthisis confirmata,* and *phthisis desperata*. Bayle, in the first decennium of the nineteenth century, added a fourth stage, *Phthisis occulta,* or *germe de la phtisie,* which corresponds to the modern pretuberculous stage, when the tubercles in the lung are too few to produce symptoms. Laennec, who was an excellent and pioneer pathologist and clinician, having invented auscultation, divided phthisis into three stages, basing his classification on anatomical grounds. He divided phthisis into: *First stage,* the accumulation of the tubercles, which betray themselves by bronchophony and dulness over the affected area; *second stage,* softening of the lesion, producing bronchial breathing, coarse rales and pectoriloquy; and *third stage,* the elimination of the softened area, leaving pulmonary excavations which may be found by careful physical exploration.

This division of phthisis into three or four stages has remained to date not only among the laity, who fear the second and third stages, but also among physicians, who are always aiming at discovering the disease in the pretuberculous stage, or at least in the first, or incipient stage. Some even maintain that the disease is curable only at this stage. That this is not always true will be shown later on.

OFFICIAL CLASSIFICATION OF THE STAGES OF PHTHISIS.

With the advance of knowledge of the clinical manifestations and the methods of recognition of the disease, the stages into which phthisis is divided remained practically the same. They have only been more

[1] Berl. klin. Wchnschr., 1911, **47**, 1.

exactly defined. In Germany the classifications of Turban and Gerhardt have gained wide acceptance, while in this country the American Sanatorium Association, and the National Tuberculosis Association have adopted the following classification, which of late has also been in vogue in most Anglo-Saxon countries:

Incipient.—Slight initial lesion in the form of infiltration limited to the apex of one or both lungs or a small part of one lobe. No tuberculous complications. Slight or no constitutional symptoms (particularly including gastric or intestinal disturbance or rapid loss of weight).

Expectoration usually small in amount or absent.

Tubercle bacilli may be present or absent.

Moderately Advanced.—No marked impairment of function, either local or constitutional. Localized consolidation moderate in extent with little or no evidence of destruction of tissue or disseminated fibroid deposits. No serious complications.

Far Advanced.—Marked impairment of function, local and constitutional. Localized consolidation intense, or disseminated areas of softening, or serious complications.

Shortcomings of the Official Classifications.—If the object of this classification is to define the prognosis of phthisis, it fails utterly. A patient with a "slight initial lesion in the form of an infiltration of the apex" has not always a greater expectation of life than one having "marked local impairment of function and extensive destruction of tissue." In fact, in acute miliary tuberculosis of the lungs the lesion is so slight that it often cannot be localized during life. On the other hand, many cases of phthisis with extensive excavations have a better outlook, at least as regards duration of life, and even as regards regaining efficiency, than some with limited lesions at one apex, without expectoration of tubercle bacilli, but with evidences of severe toxic activity. Moreover, it is clinically wrong to put into one class the incipient cases showing no fever, no tachycardia "at any time during the twenty-four hours," no gastric or intestinal disturbances, no rapid loss of weight, etc., which are evidently cases of abortive tuberculosis, if at all actively tuberculous, with those having lesions limited to one or both sides, and who do show constitutional symptoms of toxemia. The former will recover within a few months under any rational form of treatment, or spontaneously, while the latter may not, even with the most rigid institutional, climatic, dietetic, or specific treatment.

Physicians having opportunities to observe many tuberculous cases are struck with the fact that the prognosis, immediate and ultimate, does not entirely depend on the changes in the breath sounds, the presence or absence of rales, and signs of excavations in the lungs. The constitutional symptoms, such as fever, pulse-rate, presence or absence of dyspnea, gastric disturbances, and, above all, the resistance of the patient, play a greater role in the ultimate outcome of a case than the anatomical changes.

In order that a case may be considered "incipient," according to this classification, and nearly all others which have been devised, the constitutional disturbances must be slight or absent. Thus, in the definition of terms it is stated that "the impairment of health may be so slight that the patient does not look or feel sick in the ordinary sense of the word." The pulse should not exceed 90 per minute and the temperature 99.5° F., and the sputum may be negative. The physical signs consist in "slight prominence of the clavicle, lessened movement of the chest, narrowing of the apical resonance with lessened movement of the base of the lung, slight or no change in resonance, distinct or loud and harsh breathing with or without some changes in the rhythm (*i. e.*, prolonged expiration), vocal resonance possibly slightly increased; or fine or moderately coarse rales present or absent. If sputum contains tubercle bacilli, any one of these." Considering that the apex is defined as "that portion of the lung situated above the clavicle and the third vertebral spine," it is clear that the lesion must be quite limited, often of the type considered "dubious" by many competent clinicians.

All these symptoms, or absence of constitutional symptoms and signs in the chest, may be found in a large proportion of persons in all walks of life, working hard at their occupations, who, if followed for many years, are not found to develop active phthisis. People with collapse induration often show more distinct physical signs at one apex, yet they are not phthisical. In the instructions given to examiners of recruits, such individuals were ordered taken into the armies in every European country, as well as the United States.

On the other hand, a really phthisical person showing so few signs on physical exploration, but in whom the disease is pursuing an acute or subacute course, may be carried off much quicker than many with extensive involvement, but manifesting a tendency to chronicity of the process.

It cannot be denied that these three or four stages of tuberculosis are altogether arbitrary. We cannot often separate them by sharp lines of demarcation and say "this is a first stage case," or "this case is passing from the second into the third stage," etc. There are always transitional forms. There are also numerous cases showing healed lesions which at the time of activity were in the third stage, but give no more trouble—while an initial lesion in the other lung is responsible for the disease for which the patient consults the physician. Such cases, incipient in the true sense of the word, must be considered far advanced according to this classification. It is also a fact that, for phthisis to end fatally, it is not necessary that the lesion in the lung should soften and produce a cavity; caseation alone, when extending rapidly, may kill; the patient has thus not reached the third stage, yet he dies. On the other hand, we have numerous patients who, despite the fact that they have more or less extensive excavations in the lungs, are in fact in the third stage of phthisis, yet they feel well,

and are even efficient at their occupations, and when they finally die the cause may be another disease.

For these reasons some clinicians have been constrained to distinguish the various forms of phthisis met with in practice into different clinical entities. Thus, even the classification mentioned above considers acute miliary tuberculosis as a distinct disease. Other authors, like Alfred Loomis, Williams, Andrew Clark, Douglas Powell, etc., have described fibroid phthisis—which in the above classification would always be included among the advanced cases—as a distinct disease.

Many writers on this subject have gone much further and distinguished not only acute and chronic forms of the disease but have also described congenital, or hereditary and acquired forms of the disease; phthisis in arthritic, gouty, diabetic, nephritic, alcoholic, or syphilitic subjects; also according to some prominent symptoms, such as hemorrhagic, bronchitic, bronchiectatic, pleuritic phthisis. In accordance with certain etiological factors, there has been described phthisis in workers at dusty occupations, such as miners' phthisis, etc. Finally tuberculosis of the lungs in children has always been considered as presenting a different clinical picture from that in the adult; while in aged persons the symptomatology of phthisis differs from that in younger individuals.

Classification in the Present Work.—The classification of the diversity of clinical types of tuberculosis of the lungs, to be of practical value, if it is to be attempted at all, must have a prognostic value. For this reason the acute forms of the disease are to be separated into a class by themselves, as has, in fact, been done by all writers on the subject. In chronic phthisis the ultimate outcome of the disease depends mainly on the relative intensity of the two processes in the lungs, the destructive and the reparative, the former manifesting itself by caseation and softening, and the latter by the formation of fibrous tissue which limits the destructive process and even heals the lesion by cicatrization. Both processes, fibrosis and necrosis, are caused by the tubercle bacilli. And inasmuch as there are many cases in which fibrosis dominates the anatomical changes in the lungs, and the symptoms thus produced differ from those in which the caseating process predominates, it is clear that there is justification for separation of fibroid phthisis into a distinct class of the disease. This justification is fortified by the fact that the prognosis of fibroid phthisis is distinctly more favorable than that of chronic caseous phthisis, and the treatment indicated is different from that in other forms.

In common chronic phthisis we find that among the cases which have been described as "incipient" there are many which show a marked tendency to cicatrization of the lesion, spontaneously, or after some treatment for a few months. In the vast majority of cases this form of phthisis is not at all recognized and only at the autopsy some scars or calcified foci are found in the lung or pleura showing that the person had survived a tuberculous lesion. To treat these

cases in the same manner as we treat common chronic phthisis is wrong. We should, when diagnosticating a case of this kind, tell the patient that his malady is relatively trifling, and that he will recover within a few months, if he observes ordinary hygienic and dietetic rules. We can often also spare him the trouble and the economic danger of giving up his business which is usually necessary in cases of chronic phthisis. We have therefore described abortive tuberculosis as a distinct clinical type of the disease.

Most of the victims of tuberculosis who succumb to the disease, or who suffer from it for long periods of time, even if they recover, are affected with chronic phthisis. This disease is characterized by an undulating course, marked by periods of quiescence of longer or shorter duration, and interrupted by periods of acute, or subacute exacerbations. In fact, it may be stated that acute progressive phthisis, or galloping consumption, consists clinically in an acute exacerbation of the disease which is not followed by a period of quiescence. In the chronic type of the disease, proper and timely treatment may save the patient, while negligence in this regard is apt to prove disastrous. For this reason it is imperative that it should be recognized as early as possible. We have therefore divided the subject into two parts: incipient phthisis and advanced phthisis. The former, if recognized in time, and appropriate treatment applied, may often be aborted; or acute exacerbations leading to irreparable damage of the lungs and other organs and functions may be prevented. The latter, when properly cared for, may be kept in check so that acute exacerbations occur less frequently, or not at all, and cicatrization of the lesion goes on unhindered.

We also know that tuberculosis in children is anatomically, and also clinically, not of the same character as that in adults. In the former the glands, bones and joints, while in the latter the lungs, are mainly the organs which bear the brunt of the infection. Indeed, considerable harm is done to children by treating them for chronic pulmonary tuberculosis which, before the eighth year of life, they practically never have. For this reason, the disease as it occurs in infants and children merits separate description. Because in infancy the infection is usually followed by acute manifestations, while in children between two and ten years of age chronic disease of the glands occurs, we shall speak of tuberculosis in infants and tuberculosis in children.

Finally, it is now known that phthisis occurs in the aged just as frequently as in younger individuals, but that it is not recognized very often because of the peculiar symptomatology it presents. Aged consumptives, believing that they only suffer from chronic bronchitis, asthma, or pulmonary emphysema, are sources of infection to an extent not so fully appreciated as they deserve. We have therefore devoted a special chapter dealing with tuberculosis in the aged, pointing out its clinical manifestations.

While in nearly every case of pulmonary tuberculosis the pleura is

implicated in the process, more or less, there are cases in which the disease begins in the pleura and shows no tendency to spread into the pulmonary parenchyma. In others, the pleural lesion is the main one with which the patient has to cope. Moreover, it appears that the vast majority of pleurisies which had formerly been considered "idiopathic," are in reality tuberculous in character. For these reasons a book on pulmonary tuberculosis is incomplete unless a detailed account is given of tuberculosis of the pleura.

These forms of phthisis do not exhaust the subject of the clinical polymorphism of this disease. There are many other types which may be appreciated when carefully studying the cases, while quite often these types overlap one another to an extent as to render it difficult to decide to which class a case belongs. But for practical purposes these clinical types are sufficient. They assist in appreciating the course of the disease when it occurs, and give us hints for prognosis and treatment which are invaluable, and which cannot be had when pulmonary tuberculosis is considered as a single clinical entity.

We shall therefore describe phthisis under the following headings:

1. *Chronic phthisis, incipient stage.*
2. *Chronic phthisis, advanced stage.*
3. *Abortive pulmonary tuberculosis.*
4. *Fibroid phthisis.*
5. *Acute forms of phthisis.*
6. *Pulmonary tuberculosis in children.*
7. *Phthisis in the aged.*
8. *Tuberculosis of the pleura.*

CHAPTER XIX.

CHRONIC PHTHISIS, INCIPIENT STAGE.

INCIPIENT PHTHISIS.

Onset.—A lay writer,[1] describing his own, subsequently fatal, case of phthisis, in speaking of his "initiation into T. B.," says: "The entrances are innumerable, however sole the exit. Indeed, the initiation varies so widely that one would not be far wrong in saying that it is never twice the same. Yet many initiations have certain features in common, and in a general way it may be said that all belong to one of two great classes—the sudden and the protracted." No physician, however extensive his experience with phthisis, could do more justice to the subject, or make a better generalization of the various ways in which phthisis is likely to begin.

A sudden or abrupt onset of phthisis is infrequent, but it does occur. We meet with patients who have been in the best of health; have no ascertainable hereditary taint; have not come into immediate or intimate contact with a consumptive, so far as they can remember; have not overworked, not suffered from exposure, but they suddenly begin to cough, lose weight, have fever, feel tired at the least exertion, and a careful physical examination reveals a small, but progressive lesion at one apex. We meet with others who, without any premonitory symptoms, without any exciting cause, suddenly perceive a warm sensation in the throat, cough, and bring up a mouthful of blood. The hemoptysis may be scanty or copious, but the signs elicited while examining the chest leave no doubt that it is derived from a pulmonary lesion, and the subsequent course of the disease proves conclusively that we are dealing with phthisis. Still others, after an indiscreet exposure to the vicissitudes of the weather, or after a cold bath to which they are not accustomed, begin to cough and are treated for a "cold," "grippe," etc., for some time. But the symptoms fail to ameliorate in spite of careful treatment, when one day a careful examination of the chest shows a distinct lesion, or a bacteriological examination of the sputum reveals the presence of tubercle bacilli. In some, exposure may bring on an attack of pleurisy, dry or with effusion, the subsequent course of which is distinctly that of phthisis.

But in a large proportion of cases the disease develops slowly, insidiously—the initiation is protracted, as our lay friend said. For months, perhaps a year or two, the patient has not been well. He has been "sub-

[1] Atlantic Monthly, June, 1914, **113**, 747.

ject to colds," and in autumn or winter he passed through one or more attacks of "grippe," bronchitis, etc., with cough, expectoration, fever, malaise, etc., but he soon recuperated and worked more or less efficiently at his vocation. Finally one attack sticks and he does not improve, notwithstanding the remedies which were formerly effective.

In young men the symptoms which we are apt to label as "neurasthenia," may have been present for a year, two, or more. What was most annoying, and could not be relieved by the usual treatment instituted, was the languor, the tired feeling which overwhelmed the patient before his day's work was at an end. He may be complaining of cardiac palpitation, indefinite pains in the chest, some cough in the morning, etc. But on the whole he considered himself "run down," and sadly in need of a rest.

In young women the subjective and objective symptoms of chlorosis may have been present for months or years. An examination of the blood has, indeed, shown a low percentage of hemoglobin, and large doses of some iron preparation have been used. Some have had irregularities in the menstrual function, perhaps amenorrhea for several months, and even this was attributed to the anemia. But then they begin to cough; and the cough persists in spite of treatment, when an examination of the chest, or of the sputum, tells the story.

Others have been "run down" from overwork, physical or mental, for a long time till it is discovered that the cause of their debility is located in the lungs. In many patients the symptoms of dyspepsia are so pronounced as to preclude a careful examination of the chest and they are treated for a long time for "stomach trouble."

This does not exhaust the variety of symptoms which may slowly, but surely, usher in phthisis. But numerous as they are, they have certain features in common which characterize phthisis in the vast majority of cases, so that if this disease is only borne in mind—and it should, considering its great prevalence—more really incipient cases would be recognized than at present. These clinical phenomena will now be discussed.

Symptoms.—Practically all patients with incipient phthisis cough at a very early stage of the disease, and the cases without cough, which have been mentioned by various authors, are rare clinical phenomena, at least they are exceedingly rare among persons under fifty years of age, and may be disregarded. It was already stated that patients who claim that they do not cough are usually individuals who overlook a mild cough, but those around them are apt to notice that they do, and in doubtful cases it is advisable to inquire among those who live with the patient.

A person who never coughed, but after a "cold" coughs for more than two weeks, should excite interest and careful study. If he vomits after fits of coughing, tuberculosis is to be strongly suspected. Paroxysmal coughing spells are also apt to take place during the night

and keep the patient awake. Very little expectoration is apt to be brought up at this period—at most some viscid mucus which contains no or few tubercle bacilli, nor elastic tissue, though animal inoculation may be effective in disclosing the tuberculous nature of the trouble.

Languor is a constant symptom at a very early stage—the patient feels tired in the morning at rising, but recuperates after working for a few hours. But in the later part of the afternoon he feels fatigued, often drowsy, inclined to sleep. It is this tired feeling which is to be held responsible for the fact that so many patients are erroneously treated for neurasthenia and psychasthenia, or for a "nervous breakdown," for a long time until the true nature of the disease is finally ascertained.

Anorexia is an inconstant and variable symptom of incipient phthisis. In some, especially in youthful subjects, it is very frequent and, coupled with anemia, constipation, etc., is the cause why so many are treated for chlorosis, gastritis, etc. There are many cases in which the appetite is well retained and, when not "dieted" with a view of improving nutrition and digestion, but urged to eat well and plenty of the foods they are accustomed to eat, they do not lose in weight, but may gain, even when the process in the lung goes on actively.

But in the majority of cases a persistent *loss of weight* is noted at this period. In some it is slow, only one pound per week on the average, while in others it is more rapid and during the first two months fifteen to twenty pounds may be lost.

The activity of the process is best estimated by the *fever*, which is never absent. It may be slight, only 1° elevation in the afternoon, but it can be found in every case by the judicious use of the thermometer. A subnormal temperature during the early morning hours, best looked for by taking it per rectum before the patient leaves his bed, is very frequently observed, and of immense diagnostic significance. In many the fever subsides when the patient is kept in bed for a couple of days but reappears as soon as some exercise is allowed. In those with an apparently normal temperature, fever may be provoked by walking or any other form of exercise, as was already discussed in detail (see page 218). In women, the fever may appear only during the menstrual period, or a few days before.

In a large number of cases pyrexia is considerable even at this early stage, up to 102° or 103° F. in the afternoon and evening and, measured by comparison with the subnormal temperature in the early morning hours, it is quite high. The "reversed type" of fever, with a rise in the morning, is occasionally seen.

A significant diagnostic point is that *with high fever the patient may not be prostrated as is the case with adults who have fever due to other causes.* Moreover, the patient may have a fair, even a good appetite, despite the fact that the thermometer registers 102° or 103° F., which is very rare in fevers due to other causes. During the

afternoon access of the fever, the patient, otherwise pale, becomes flushed, often only one cheek is red, he is tired, and disinclined to work. But he may keep on working, as was already stated.

Nightsweats make their appearance in a large proportion of cases at this stage. In some they are slight, while in others I have met with profuse nightsweats during the first two weeks of active symptoms. They perspire also at the least exertion or excitement, and during a medical examination it is not rare to see large drops of sweat dribbling down the sides of the chest from the axillæ.

A constant accompaniment of fever in incipient cases is *tachycardia*. A case of active phthisis with a pulse-rate below 80 per minute is exceedingly rare. In some the heart action is so rapid that they are treated for heart disease, or for hyperthyroidism in case the thyroid is enlarged, which is not rare, especially in youthful individuals. While in the early morning after a refreshing sleep the pulse-rate may be normal, the least exertion or excitement will raise it up to 90, 100 or more. *Instability of the pulse* is characteristic of phthisis. In youthful subjects the tachycardia is apt to be more pronounced than in persons over twenty-five years of age. The *blood-pressure* is low and a registration less than 80 mm. of mercury is quite common.

Symptoms referable to the respiratory system may not be seen at this stage, excepting the cough and, at times, the *intermittent hoarseness*, which is usually due to a laryngeal catarrh, or pressure on the laryngeal nerve, and hardly ever to infiltration of the larynx. At times we see patients who suffer from more or less pronounced pains in the chest, especially under the scapula, or in the shoulder.

Hemoptysis is quite frequent at this stage. As was already stated, statistics taken of large numbers of patients show that about 10 per cent. of cases begin with hemorrhage. They are the lucky ones, because this clears up the case, and proper measures are promptly taken. But many of these initial hemorrhages were actually preceded by a train of symptoms, such as fever, tachycardia, etc., to which the patient paid no attention. However, in about 25 per cent of cases more or less blood-spitting occurs at the time the disease is recognized. It may be only blood-tinged sputum, a mouthful or two of blood, or even a profuse hemorrhage. It will bear repetition that these initial hemorrhages are practically never fatal.

Physical Signs.—With any or all of these symptoms a diagnosis of incipient tuberculosis should not be made unless physical exploration of the chest discloses a localized lesion in the lungs.

Inspection.—Inspection yields excellent diagnostic criteria in most cases at this early stage. Inasmuch as most of the incipient cases are really recrudescences of old quiescent lesions in many instances dating back to childhood, we find in many atrophy of the muscles over the site of the lesion. The sternocleidomastoid, the scaleni and trapezius, etc., may be smaller than those on the opposite side, and softer, or even flabby to the touch. This is more important to look for than

the form of the chest, which may be normal, flat, rachitic, etc., without influencing the diagnosis. With the atrophy of the muscles there is usually seen a slight shoulder-droop and an excavation of the supraclavicular or supraspinous fossa, or at least some flattening and defective motion or lagging of the part of the chest harboring the lesion. This asymmetry, flattening and lagging, is very easy to detect if carefully looked for and is, when found, of immense diagnostic importance, provided occupational influences are excluded.

In more recent lesions, or when an old and perhaps a healed lesion exists in one side, but the outbreak of phthisis is due to a new lesion in the opposite side, which is very frequent, we find the muscles over the site of the active new infiltration are tense and rigid, standing out prominently. But this is after all not very frequent, which goes to show that most of the incipient cases are really due to reawakening of old, smouldering, tuberculous processes in the lung.

Percussion.—As was already stated, there are very few cases of active incipient tuberculosis in which no signs of an infiltration can be discovered by careful and gentle percussion. We almost invariably find some airless pulmonary tissue, or shrinkage of one apex manifesting itself by a short note, or by pulmonary retraction. The height of the apex may be less than that of its mate on the opposite side, or its width may be less, as determined by Krönig's method of percussion. We may also find, though not so often as Krönig believed, that the base on the affected side is somewhat retracted. Immediate percussion of the clavicle, as was first practised by Laennec, may at times easily reveal a lesion beneath that bone.

In my own experience, percussion signs are more often found over the posterior aspects of the apices than anteriorly. While over the supraclavicular region we may find that the width of the resonant area is less than that of the other side, it is easier and less time-consuming to map out the mesial lines of demarcation between resonance and dulness in the supraspinous fossæ, and over the site of the lesion this line is usually dislocated outward.

It is also easily ascertained whether the height of the apices posteriorly shows any asymmetry. At a very early stage we find that while over the unaffected apex the resonance reaches as far as the interval between the seventh cervical and first thoracic spines, that of the affected apex is much lower. I have found these changes at times before any auscultatory signs made their appearance.

The changes in pitch, duration and intensity of the note obtained at this stage are of less significance than those of shrinkage just spoken of, and they depend too much on the personal equation of the observer to have important clinical bearings. Thus, we often find that a contracted apex is altogether hyperresonant, or even tympanitic on percussion, and by comparison the unaffected side appears to emit a defective note. The stories told in text-books about two equally competent clinicians localizing an apical lesion by percussion, and each finding

it in another side, are undoubtedly based on these facts. It is generally due to faulty interpretation of tympany caused by relaxation and hyperfunction of the lung tissue around conglomerations of tubercles, as has already been shown. The discordance may also be due to an old and cicatrized lesion on one side, while the new and active lesion is in the opposite side of the chest.

Of greater importance is respiratory percussion. The patient is asked to inspire or expire and hold his breath, and we percuss during each phase of respiration. In health the note is clearer during full and held inspiration, while over an infiltrated apex a long and held inspiration gives a duller note than found over the opposite, unaffected side.

Of the various seats of election of dulness in incipient phthisis which have been described by Lees,[1] Riviere,[2] and others, the sites I have been able to find impaired in most cases at a very early stage are the supraspinous fossæ, near the spine, and beneath and above the inner third of the clavicle. Persistent, impaired resonance in any of these places, when accompanied by constitutional symptoms of phthisis, is of diagnostic significance. Impaired resonance elicited with hooked-finger percussion between the heads of the sternocleidomastoid immediately above the clavicle on one side is very often found.

Auscultation.—It is not generally appreciated that the earliest changes in the respiratory sounds in phthisis are modifications of the inspiratory murmur, while changes in the expiratory murmur usually indicate a more or less advanced stage of the disease. At a very early period of the disease the inspiratory murmur loses its soft, breezy character and becomes rough or granular, an indication of partial stenosis of the bronchioles supplying the affected part of the lung, or unequal respiratory movement of the infiltrated lung area. In many cases the inspiratory murmur is feeble, at times even absent, over a limited area corresponding to the area of impaired resonance, while the whispered voice is transmitted clearly. But the most common sign of an early lesion is interrupted, or cog-wheel, breathing; the inspiratory sound is broken up into several parts so that it appears jerky. Either of these types of altered inspiratory murmur may be audible long before the expiratory murmur is in any way changed.

The most common seats of the changed breath sounds are posteriorly near and above the spine of the scapula, the "alarm zone" of Chauvet,[3] and rarely in front immediately beneath the inner third of the clavicle. It is located posteriorly as follows: From the space separating the spinous process of the seventh cervical from that of the first thoracic vertebræ, a line is drawn as far as the tubercle of the trapezius on the spine of the scapula. From the middle of this line, taken as a center, a circle is described with a diameter equal to

[1] British Med. Jour., 1912, **2**, 1268.
[2] Early Diagnosis of Tubercle, London, 1914, p. 25.
[3] Le monde médical, 1913, **22**, 1121; La Clinique, 1913, **8**, 437.

that of a silver dollar. The circumference of this circle forms the boundary of the "zone of alarm" (Fig. 83). When heard at any of these points during ordinary breathing, and repeatedly found on several examinations, not decreasing in intensity but on the contrary becoming more and more pronounced, rough and cog-wheel breathing are good signs of incipient infiltration of an apex, provided of course, that the constitutional symptoms show activity; otherwise they may be indications of old and cicatrized lesions. We have already stated that at times feeble breath sounds are found; they may be of a blowing or even of a bronchial character, and some crackling may be audible at the end of inspiration.

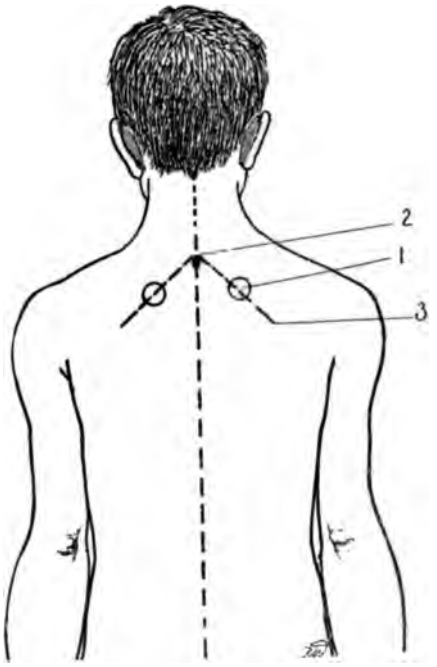


FIG. 83.—1, The "alarm zone"; 2, the space between the spinous processes of the seventh cervical and first dorsal vertebræ; 3, the tubercle of the trapezius.

Rales are not heard at all over really incipient lesions. Occasionally some sibilation is audible, but this is usually transitory and disappears after the patient takes a deep breath. At most, some dry crackling may be brought out when the patient coughs vigorously. When crepitant, and especially moist subcrepitant, rales are audible, we are dealing with an extensive lesion of some duration.

In some cases we hear at a very early stage during expiration a hemic murmur originating in the subclavian artery and ascribed to kinking of that vessel by the tuberculous infiltration, or by shrinking

lung. But it is by no means pathognomonic of phthisis because it is heard in many apparently healthy persons.

The whispered voice is very often transmitted more or less clearly over consolidated areas fo lung tissue, and when heard when the chest-piece of the stethoscope is pressed firmly over the skin of the chest, it is of the same diagnostic significance as impaired resonance, with which it usually runs parallel, as has been pointed out by Sewall.[1] But it must be emphasized that its absence does not exclude a tuberculous lesion. The voice sounds are transmitted only when the consolidated tissue is located superficially or subpleurally. When it is centrally located, screened by air-filled lung tissue, the voice sounds may be normal.

To be of diagnostic significance in early phthisis, the auscultatory signs must be localized over one apex, circumscribed, fixed and persistent for some time, and not influenced by cough and strong respiratory efforts, excepting clicks and rales which may be brought out by cough. Evanescent changes in resonance and breath sounds may be found in many apparently healthy persons. It is for this reason that those who attempt to make a final diagnosis of incipient phthisis during one examination meet with so many failures.

Elements of Diagnosis of Incipient Phthisis.—Just as the general and constitutional symptoms, such as cough, fever, tachycardia, emaciation, etc., are insufficient to decide a case till the lesion is localized in the lung, so are the signs obtained by physical exploration of the chest inadequate, even when marked, to prove that we are dealing with a case of active incipient phthisis. Only the combination of both groups of clinical data gives solid support for diagnostic inferences. A skilled diagnostician may easily diagnosticate a case of advanced phthisis by looking at the pathognomonic facies of the patient, from the history and course of the disease, or from auscultatory findings alone, and only rarely err. But *in incipient phthisis it is the correlation of all available clinical data, the history, the symptomatology and course of the disease, combined with the findings of physical exploration of the chest and bacteriological as well as roentgenological data that can be expected to clinch the diagnosis.*

The signs enumerated above—defective resonance, narrowing of the resonant areas over one apex, feeble, rough, granular or cog-wheel breathing, or even rales, may each be found in persons of excellent health, at least such as are not actively tuberculous. This is because old and healed lesions, tuberculous and others, leave traces behind them which alter permanently the air content of the pulmonary parenchyma and diagnostic methods in vogue disclose these conditions.

Sources of Errors.—I am not prepared to state that the proportion of diagnostic errors made while attempting to recognize phthisis in its very incipiency is greater than in other diseases; in fact, I am convinced

[1] Jour. Am. Med. Assn., 1913, **60**, 2027.

that it is not. But in phthisis, owing to its great prevalence and its social and economic aspects, as well as its insidious onset, the opportunities for making mistakes are immense. It is for this reason that the sources of error must be emphasized.

Bias is more often a source of error in phthisis than in any other disease. Especially is this the case when there is a history of exposure to infection. To my mind this is one of the greatest fallacies we have to cope with. It must always be remembered that in large industrial cities everyone is exposed to infection and is, in fact, infected with tubercle bacilli before he passes his fifteenth year. On the other hand, marital phthisis is less frequent than would be expected if every adult exposed to tuberculosis would invariably or in most cases become phthisical. Excepting in young children a case must therefore be judged on its clinical manifestations and not on the fact that the patient came into contact with a consumptive.

Tubercle Bacilli.— *The diagnosis of phthisis is clinched by the finding of tubercle bacilli in the sputum, but the disease is not at all excluded by negative bacteriological findings.* Unfortunately, too many wait rather long for the bacilli, thus losing valuable time which often cannot be reclaimed by any known means. Phthisis begins as an infiltration, and only when softening has taken place and the products of tissue disintegration are eliminated through a bronchus can tubercle bacilli be found in the sputum. Under the circumstances, *waiting for tubercle bacilli to make their appearance in the sputum is just as hazardous as waiting for pus to make its appearance through a fistula or sinus before making a diagnosis of a tuberculous joint.*

In very rare instances there are errors of quite a different nature. *Tubercle bacilli may be found in the sputum of persons who are not actively tuberculous.* Of course, from the practical standpoint tubercle bacilli in sputum are an indication that they are in all probability derived from a tuberculous lesion in the lower respiratory tract. But in New York City we meet with numerous persons who have reports from some private, as well as from the municipal laboratory, stating that the sputum of the bearer had been examined and found to contain tubercle bacilli. Yet, without any treatment or special care, they have kept at work for years and felt well. Indeed, many cases are admitted to sanatoriums solely on the strength of positive sputum findings, to be declared non-tuberculous after careful observation.

The reasons for this anomaly are to be sought for in several facts which have not been emphasized as strongly as they deserve. I have no doubt that in busy laboratories mistakes are liable to happen in handling the sputum bottles, in numbering the slides, or while entering the findings in the reports. In banks, where the clerks are just as careful as laboratory workers, mistakes occur at times. Even conceding that the number of such mistakes is comparatively negligible, in the individual case it may count very much.

We have already spoken of the acid-fast rods which simulate tubercle

bacilli and which are found in butter and milk, on graminacea, in the soil, in dung and manure, and even in tap water supplied through metal pipes. These bacilli are dead, or non-pathogenic to guinea-pigs, but they give the usual staining reactions. Then we may have the smegma bacilli which have been mistaken for tubercle bacilli and thus have led to erroneous diagnosis and extirpation of healthy kidneys. There are also the acid-fast lepra bacilli, the microörganisms which greatly resemble them and are found in the secretion of the mucous membrane of the nose, also the acid-fast rods found in the saliva and the secretions in cases of bronchitis and pulmonary gangrene. L. Napoleon Boston[1] found acid-fast bacilli in patients suffering from acute colds and influenza, and disappearing during convalescence. But most of these microörganisms are difficult to differentiate from tubercle bacilli microscopically, through culture and animal inoculation.

It has recently been found that the spores of lycopodium are acid-fast, so that persons taking pills covered by that substance may impart some of it to the sputum and thus lead to error.

There is a possibility that the acid-fast rods or specks found in the sputum may not have been there before it left the bronchial tubes and trachea, but got into the sputum while it was passing through the pharynx, mouth or lips, especially in persons living in houses inhabited by careless consumptives. I have repeatedly observed this to be a fact in consorts of tuberculous patients. Tubercle bacilli are found in the sputum—usually saliva—but they keep up in good health. It is also important to mention that ordinary smear preparations are less likely to lead to errors of this sort than the antiformin method.

To be sure, the most reliable sign of phthisis is tubercle bacilli in the sputum, and I do not at all intend to underestimate its far-reaching significance. Statistically, the chances of error are undoubtedly insignificant, and a laboratory may be proud that among several thousands of specimens only half a dozen mistakes have been made. But the practising physician does not treat his patient statistically. In the individual case it is well to bear in mind the possibility of errors of this kind, especially in cases in which the disease does not pursue the course expected in some form of phthisis.

Roentgenography.—From the facts presented in Chapter XVII it is clear that roentgenography is of but little value in the diagnosis of truly incipient lesions in the lungs. Infiltrations, even when extensive, do not cast any characteristic shadows on the plate; the slight opacities they do produce are insufficient in themselves to clinch a diagnosis, or even to localize the active lesion, considering that there is hardly a chest in which opacities of various kinds, engendered by a multiplicity of causes, may not be seen. When the apical opacities are distinct the case is no more "incipient," because they represent either caseated areas of lung tissue or, more commonly, fibrosis and calcification of

[1] Interstate Med. Jour., 1914, **21**, 330.

pulmonary parenchyma and glands. But these processes, fibrosis and calcification, are indications of healing and may not at all be the cause of the symptoms. Peribronchial tuberculosis, which some roentgenographers describe in great detail, usually represents glandular fibrosis, calcification, etc., and bloodvessels, bronchi, and other structures superimposed and crossing each other in the region of the hilum. While some of these opacities undoubtedly represent tuberculous changes, these lesions are healed, as a rule. It is commonly the parenchymatous lesion in the apex which gives trouble in really incipient cases, and these we should like to detect as early as possible, but they permit the rays to pass unhindered. For these reasons we find that the lesion, easily localized by one well versed in physical diagnosis, is not detected by those who rely solely on roentgen findings.

These sad disappointments have led many clinicians of late to rely on the clinical aspects of the case, physical exploration of the chest, the history and symptomatology, etc., in their efforts at detecting tuberculosis as early as possible. It has been our rule never to make a diagnosis of incipient tuberculosis merely because there are positive roentgen findings when the constitutional symptoms are not in agreement. Conversely, when the constitutional symptoms are clear cut, and physical exploration of the chest points to a lesion in an apex, the diagnosis is unhesitatingly made even though the roentgen findings are negative, which is very often the case in instances with positive sputum. Exceptions are cases giving a history of several attacks of abortive tuberculosis, which may or may not have been diagnosed at the time of their occurrence, and these attacks have left traces behind which reveal themselves on the plate.

Serious errors are at times committed by those who rely solely on roentgen findings. A patient with sclerosis of the apex which is perfectly healed acquires some mild ailment which is characterized by cough, fever, etc., is sent to a radiographer who pronounces him tuberculous, and appropriate costly and prolonged treatment is instituted. Therapeutic pneumothorax is induced in the side of the chest where the opacities on the plate are more extensive and pronounced. But these opacities represent healed lesions, while the slight roentgen changes in the opposite side really represent the active and progressive lesion. The results in such cases are often disastrous.

For these reasons no diagnosis should be made of incipient phthisis relying solely on the roentgen findings. Clinical observation should be given more attention if we are to avoid errors of omission or commission.

The Tuberculin Tests.—The changed reactivity to tuberculin which is observed in organisms infected with tubercle bacilli, manifesting itself mainly by hypersensitiveness to that agent, has been applied in the diagnosis of doubtful cases, especially in sanatoriums. When first introduced it was heralded as specific and it was asserted that finally a positive and uncontrovertible test had been found which decides whether or not an individual is suffering from active tuberculosis.

For diagnostic purposes, tuberculin is applied in various ways. It is introduced directly into the circulation by the subcutaneous method; into the lymph spaces by the cutaneous method, or applied to mucous membranes for normal absorption by the conjunctival method. It has thus been applied to the skin, mucous membrane, and subcutaneously. The subcutaneous application produces general and constitutional symptoms of tuberculin intoxication, while the others, as a rule, produce local effects.

Clinically the following reactions are evoked by the tuberculin test.

1. A *general reaction*, manifesting itself after the subcutaneous injection of tuberculin by fever, chilliness, malaise, headache, backache, etc.

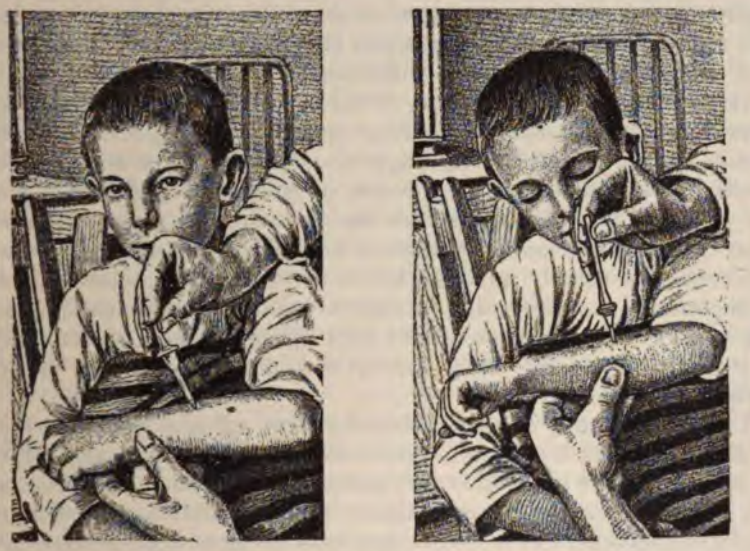


FIG. 84.—The application of the cutaneous tuberculin test.

2. A *focal reaction*, consisting in congestive and inflammatory phenomena in the neighborhood of the tuberculous lesion.

3. *Local reactions*, hyperemia and inflammatory phenomena at the site of the tuberculin application. Of these there are: (*a*) The cutaneous reaction of von Pirquet and several of its modifications; (*b*) mucous membrane reactions, such as the ophthalmo-reaction of Calmette and Wolff-Eisner, etc., and many others which have been discarded for valid reasons.

The Cutaneous Tuberculin Test.—This is the simplest and unquestionably *the* harmless method of application of tuberculin for diagnostic purposes. It is usually performed on the inner surface of the forearm, though any part of the body may do; but it appears that the skin of the trunk is not so sensitive as that of the forearm and thigh.

The skin is cleaned with alcohol or ether and a drop of pure tuberculin is applied. A suitable instrument is then used to make two abrasions, one about two inches away from the spot where the tuberculin has been applied, and the other over the tuberculin. The instrument devised by von Pirquet may be used. It consists of a heavy handle with a spade-like platinum end which is more or less sharp and used for the purpose of scratching or boring a cup-like depression in the skin. It is important that bleeding should not be caused, but only the superficial layer of the skin is scraped away, so as to open the lymph spaces and thus favor absorption of the tuberculin. A needle may be used for the purpose or even the point of a scalpel, making one or two parallel incisions through the superficial layer of the skin. I have found it just as effective to make the abrasion first and then apply the tuberculin with a toothpick, rubbing it vigorously. After five minutes the excess of tuberculin is wiped away with some cotton and the patient allowed to go without any dressing.

If the test turns out negative, it will be seen that twenty-four hours later the two abrasions either heal in the same manner, or when a scab is formed it is of the same appearance on both abrasions. When positive, the control appears healed, or showing a slight scab, while the abrasion to which tuberculin has been applied shows an inflammatory infiltration manifesting itself as a slightly elevated, red papule. This reaction usually appears twelve to twenty-four hours after the application of the tuberculin; on rare occasions it is premature, appearing within four to six hours, and may disappear soon, or remain for days; or it may be late in appearing; even a delay of a week has been observed in rare cases.

The reaction may be slight, showing some redness with infiltration, or a more extensive area of redness with an appreciably raised papule. In some cases the red area is very extensive, simulating erysipelas, and the papule is very elevated. Quite often the first test results in a negative outcome, but a second application, about a week later, gives positive results. It is therefore advisable to repeat the test two or three times before pronouncing it unequivocally negative.

These "secondary" reactions are usually very intense, although the first application was negative. It has also been noted that the tuberculin sensitiveness is often increased by a second or third inoculation and the area at which the first inoculation was made also reacts. Attempts to utilize these facts for diagnostic purposes have not been encouraging.

Significance of the Cutaneous Tuberculin Reaction.—Clinical experience has shown conclusively that persons who have at any time been infected with tubercle bacilli react to the cutaneous tuberculin test; experimental investigations have confirmed it. It is immaterial whether the infection is followed by clinical manifestations of disease or not; whether the tuberculous lesion is active or quiescent, the result is the same. It appears to me, however, that we do not have sufficient

evidence for a conclusion as to the question how long after a lesion has healed does the skin remain sensitive to tuberculin. Assuming that no tuberculous lesion ever heals perfectly, which has not yet been proved, we accept that even clinically healed lesions act in this way.

Newborn infants never react to tuberculin, but when living in tubercle-laden surroundings they soon show the hypersensitiveness, as was already shown (page 70). Inasmuch as over 90 per cent of humanity have been infected before reaching the twentieth year of life, we find that many show positive reactions to tuberculin.

It is thus clear that *for clinical purposes, when we look for evidences of active phthisis, this test is of little value, because it shows not only those who suffer from active tuberculosis, but also such as have latent or healed lesions.* Moreover, it is often negative in rapidly progressing pulmonary tuberculosis, in tuberculous meningitis, in acute miliary tuberculosis and also in the terminal stages of chronic phthisis, when the formation of antibodies is slackened or abolished. It has also been found negative in the presence of other infectious diseases, like measles, scarlet fever, diphtheria, influenza, etc., in some cases of pneumonia, and often during pregnancy. In a certain number of cases of undoubted phthisis the cutaneous reaction was found negative without any assignable reason; von Pirquet estimated it at from 2 to 4 per cent, but in my experience it is more than double that proportion.

After many years of experience with this test it was concluded by most authors that a *positive cutaneous reaction is of clinical value only in children, and that the younger the child, the more its clinical significance.* But from more extensive experience it appears that it is also unreliable in children. From personal experience I am inclined to the conclusion that *children between three and fifteen years of age with a positive tuberculin reaction are not necessarily doomed to develop active phthisis*; I have even observed many infants under two years of age grow into healthy children in spite of the positive outcome of the test, and the statement of some authors to the effect that an infant under one year showing a positive cutaneous reaction will not survive a year is negatived by the many infants I observed and reported elsewhere,[1] who have thrived despite the fact that during the first six months of their life the reaction was positive.

Specificity of the Test.—It appears that from the scientific standpoint *the specificity of the test has not been proved to the satisfaction of all,* as has already been shown. Autopsy findings by Ganghofner, Radziejewski, Behrend, Bruckner, Reüschel, and many others show that there are cases in which the test was positive, yet no tuberculous lesions were found at the autopsy, and the reverse. Experimentally the evidence is in the same direction (see p. 38).

It has also been found that tuberculin is not the only substance capable of producing a positive skin reaction in tuberculous indi-

[1] See A Study of the Child in the Tuberculous Milieu, Arch. of Pediat., 1914, **31**, 96, 197; 1915, **32**, 20.

viduals, but that other toxins when inoculated into the skin often produce changes which are akin to the tuberculin reaction. Rolly[1] found that the skin reacted when inoculated with the toxins of dysentery, typhoid, paratyphoid, pyocyaneus, cholera, etc. Just as with tuberculin, these toxins were always negative in very young infants, and in children suffering from acute infectious diseases, as scarlet fever, measles, etc., becoming positive during convalescence. The controls, performed with carbol-glycerin, were always negative. In short, these non-tuberculous toxins showed all the characteristics of tuberculin when inoculated into the human skin. That any or all of these toxins acted in a specific manner may be ruled out because, with the exception of tuberculosis, the individuals tested never suffered from typhoid, paratyphoid, cholera, diphtheria or pyocyaneous sepsis. Tenzer[2] obtained skin reactions indistinguishable from those of the von Pirquet test with cholera vaccine and with a mixture of peptoalbumoses, in persons in whom the tuberculin test was positive.

From these experiments, as well as from those performed by Sorgo,[3] it appears that tuberculous individuals react with a specific intensity to tuberculin and to other toxins, thus indicating that it is mainly due to hypersensitiveness of the skin. The assumption that the skin of the tuberculous is endowed with a specific allergy to tuberculin alone is thereby disproved. The allergy is evidently a cutaneous hypersensitiveness to the action of toxins in general. Hamburger,[4] one of the most authoritative champions of the specificity of the tuberculin test, after inoculating tuberculous patients with substances similar to those with which tuberculin is prepared (glycerin, bouillon, extractives, salts, etc.), became convinced that the cutaneous reaction is due more to the latter substances than to the tuberculin, which acts merely as a skin irritant.

We are therefore justified in concluding that we are far from having sufficient and satisfactory information to speak with certainty about the cutaneous tuberculin test and its underlying causes, and from the theoretical standpoint its specificity has not been proved conclusively.

However, for demographers the test is important in showing the wide dissemination of tuberculous infection among civilized humanity, though the same results could be also obtained with substances other than tuberculin. In children it shows whether they have been infected with tuberculosis, and *in infants it even points to active tuberculosis; but in adults it is of no clinical value at all.*

The various modifications of the cutaneous tuberculin tests are not superior to the von Pirquet method. The Moro test, consisting in rubbing tuberculin ointment into the skin, is of less value than the one described above. The percutaneous, the quantitative cutaneous test,

[1] München. med. Wchnschr., 1911, **58**, 1285.
[2] Monatschr. f. Kinderheilk., 1911, **10**, 131.
[3] Deutsch. med. Wchnschr., 1911, **37**, 1015.
[4] Die Tuberkulose des Kindesalter, p. 37.

etc., offer no advantages over the von Pirquet test, which is after all the simplest and most reliable.

The Conjunctival Reaction.—The conjunctival reaction, invented by Calmette and Wolff-Eisner, is made by instilling into the conjunctiva, with an ordinary eye-dropper, one drop of a 1 per cent solution of tuberculin. The reaction appears within twelve hours and reaches its optimum in twenty-four hours, producing redness of the conjunctiva, and when the reaction is intense, the redness is more pronounced and there is also injection of the vessels of the eyeball and more or less well-marked secretion of mucus. It may last for two or three days. Of course, in estimating the effects of the tuberculin, comparison is made with the other eye.

Among clinically non-tuberculous persons, from 10 to 25 per cent react, while among those who are evidently tuberculous, between 50 and 75 per cent show a reaction with this test. It has been practically discarded of late because in many cases quite troublesome inflammatory phenomena appeared in the tested eye. In one of my cases the inflammation was so severe, persisting for three months, that I have ever since hesitated in applying it. Bandelier and Röpke state that experiments on animals have shown that this test is unreliable in cases of human phthisis, since the reaction may be negative in spite of the presence of active tuberculosis unless 10 per cent solution of tuberculin is used, and this should not be done when dealing with human eyes.

The Subcutaneous Tuberculin Test.—This is the test preferred by most of those who have confidence in the diagnostic value of tuberculin in doubtful cases. It is claimed that it is not only reliable in deciding whether the patient has ever been infected with tubercle bacilli, but also in showing whether the disease is active and that in many cases it even shows the area involved at the time the test is made by the so-called "focal reaction."

Of the various ways in which it is performed, the following is the simplest and gives the same results as any that has been devised:

For twenty-four hours the temperature of the patient is taken every three hours and carefully recorded. Inquiries are made as to the subjective symptoms, especially pains in the chest, headache, cough, expectoration, etc. An injection of 0.1 mg. of tuberculin is then made subcutaneously in the region of the back below the angle of the scapula or any other place. Of course, all antiseptic precautions are to be rigidly observed and the skin washed with alcohol or ether. In case no reaction appears within forty-eight hours, a second injection is made with the same amount of tuberculin, while some increase it to 1 mg. This dose is again increased in case no reaction follows to 5 mg. and even to 10 mg. in case the test proves negative and a fourth injection is given. Of course, in children smaller doses are used.

The Reaction.—Usually between ten to twelve hours, rarely between six to eight hours, in case the reaction is positive, constitutional, local, and focal symptoms make their appearance. Some say that it may

be delayed as long as forty-eight to seventy-two hours, but this must be very rare; I have never encountered it. Of the constitutional symptoms, fever is the most constant and reliable. The temperature begins to rise six to twelve hours after the injection, reaching 100° to 102° F., and in those showing a severe reaction, it may even go up to 104° F., and I have seen several cases in which it was higher. There are usually constitutional symptoms of hyperthermia—headache, backache, pains in the joints, weakness, malaise and, in some cases, nausea and vomiting. Rarely the prostration is very pronounced, while in others it may be slight, or even absent, irrespective of the degree of fever. These symptoms usually subside within twenty-four to forty-eight hours and only rarely last longer.

At the site of the injection the local reaction manifests itself in tenderness or even pain, redness, and swelling, which may be small—only about 1 cm.—but in some cases the infiltration is as large as a hen's egg. Lymphangitis and enlargement of the regional lymphatic glands may occur.

The so-called "focal reaction" is very rarely observed in phthisis. It is said to consist in congestion of the lesion in the lung, an increase in number and consonance of the rales, a change in the breath sounds and extension of the dull areas, accompanied by an increase in the cough and expectoration. Tubercle bacilli hitherto absent from the sputum may now be found. My own experience leads me to the conviction that this focal reaction is very unreliable. It occurs but rarely, and when we recall that in phthisis the physical signs change so often, and that a skilful clinician one day finds signs in one side and the next day in another without tuberculin injections, we may always suspect that the focal reaction is not necessarily a result of the tuberculin injection; at least its inconstancy should lead us to this conclusion.

Clinical Value of the Test.—The object of the test is to clear up doubtful cases in which there are symptoms and signs pointing to active phthisis but which are not convincing to clinch the diagnosis. In such cases the advocates of the test claim that a positive reaction decides in favor of active disease, while a negative outcome decisively excludes it. It has been used mostly in sanatoriums for these purposes.

Careful analysis of the conditions under which this test is negative or positive shows that it is hardly of greater reliability than the cutaneous or conjunctival test. Investigations by Franz,[1] Hamman and Wolman,[2] Beck,[3] and many others show that it may be positive in healthy persons who do not develop phthisis subsequently. The experience of all who have applied this test to large numbers of actually or apparently non-tuberculous individuals is the same as that of Franz, Hamman and Wolman, Beck, etc. *It is always found that between* 40

[1] Wien. klin. Wchnschr., 1909, **22**, 991.
[2] Tuberculin in Diagnosis and Treatment, New York, 1912.
[3] Deutsch. med. Wchnschr., 1899, **25**, 137.

and 60 per cent of humanity react to the subcutaneous tuberculin test, providing it is repeated with ascending doses three or four times.

Specificity of the Test.—We have already mentioned that many non-tuberculous substances have a toxic action on the organism infected with tubercle bacilli. Thus, according to experiments by Mettetal,[1] Preisich and Heim,[2] Petruschky,[3] and many others, nucleins, blood-serum, testicular extract from healthy animals, culture-free bouillon, and other foreign albumoses, when injected into tuberculous persons, may provoke reactions not unlike the general reaction of tuberculin. More recently the parenteral injection of milk has been found to produce a reaction which cannot be distinguished from that produced by tuberculin. It appears that the tuberculin reaction is part and parcel of the hypersensitiveness of the infected organism to foreign proteins of any kind, tuberculous and non-tuberculous (see p. 38).

Diagnostic Value.—Considering that the subcutaneous tuberculin test discloses latent infection, as well as active tuberculous disease, its diagnostic value is limited, bearing in mind that over 90 per cent of humanity have been infected at some period of their lives. What we look for is active disease and when the test also shows those who are not phthisical, its value in diagnosis is limited indeed.

"A positive tuberculin reaction," say Hamman and Wolman, "is merely confirmatory evidence and never decides with certainty an otherwise doubtful diagnosis. Indeed we feel that caution is decidedly in place not to lay too much emphasis upon a positive reaction, for if a patient is suffering from symptoms which may be accounted for by a number of different conditions, and by applying the test we admit such uncertainty, a positive reaction does not impel the conclusion that these symptoms are due to tuberculosis. If such a large percentage of healthy individuals harbor clinically unimportant tuberculous lesions, a certain proportion of those suspected of having tuberculosis must likewise harbor them, though the symptoms that attract our attention may be due to some other disease." With this view the present writer agrees entirely. How far the tuberculin test has been discarded as a diagnostic agent is seen from the fact that in none of the armies engaged in the World War had it been adopted as a test for active tuberculous disease, though strong efforts were made to weed out tuberculous persons from the service.

Dangers of the Test and Contraindications.—The subcutaneous tuberculin test is not without dangers. When carelessly performed with excessive doses, latent or quiescent lesions may be flared up into activity. L. Rabinowitsch,[4] Bacmeister,[5] Leo Kessel,[6] and others have shown that living and virulent tubercle bacilli may appear in

[1] Valeur de la tuberculine dans le diagnostic de la tuberculose de la premiere enfance, Thèse de Paris, 1900.
[2] Zentralbl. f. Bakteriol., 1902, **31,** 712.
[3] Ergebn. d. inn. Med. u. Kinderheilk., 1912, **9,** 557.
[4] Berl. klin. Wchnschr., 1913, 1. [5] München. med. Wchnschr., 1913, **60,** 343.
[6] Am. Jour. Med. Sci., 1915, **150,** 337.

the blood after an injection of tuberculin. In some cases it has been observed that hemoptysis is provoked by the test, and all agree that it must not be given during, or soon after, a pulmonary hemorrhage. In general the reaction consists essentially in a transient toxic injury to the body, and the nervous system bears the brunt of the traumatism.

It has also been found dangerous in cases of heart disease, arteriosclerosis, nephritis, diabetes, etc. In epileptics it has been observed that the reaction may provoke convulsions. Even Bandelier and Röpke say that it is contraindicated when miliary tuberculosis is suspected, "since its downward course might be accelerated." Sahli,[1] who uses tuberculin for therapeutic purposes extensively, says: "The use of tuberculin for diagnostic purposes ought to be condemned. It is unreliable both positively and negatively. Diagnostic injections are dangerous."

The Complement-fixation Test.—Quite recently the complement-fixation test on the lines of the well-known Wassermann reaction for syphilis has been applied in the diagnosis of tuberculosis. It has been studied by Besredka and Manoukhine,[2] Calmette and Massol,[3] Debains and Jupille[4] in France, and in England by James McIntosh, Paul Fildes,[5] J. A. D. Radcliffe and Edward Glover.[6] In this country, J. Bronfenbrenner,[7] A. M. Stimson,[8] Charles F. Craig,[9] H. R. Miller, and others have reported good results with this test.

But so far the results appear to be conflicting in certain points, so that further careful research combined with clinical observations are necessary before deciding on the specificity and clinical applicability of the test in general practice. The main difficulty is evidently the fact that different authors have used different antigens. Besredka used one prepared from egg-broth cultures of tubercle bacilli; Radcliffe used a freshly prepared unsterilized emulsion of saline solution of living tubercle bacilli grown on glycerin-egg medium; Hammer used an alcoholic extract of tuberculous tissue to which was added a certain amount of old tuberculin; Stimson and Bronfenbrenner use Besredka's antigen; Craig's antigen consists in an extract of several strains of human tubercle bacilli prepared by a special method. It is thus clear that with so many different methods the results are hardly comparable. Moreover, as McIntosh points out, Besredka's antigen, cannot be considered absolutely specific since Inman and Küss and Leredde and Rubinstein found that non-tuberculous syphilitics gave the reaction frequently. Even if the explanation that it is due to the lipoids derived from the egg constituents of the medium,

[1] Fifth Confer. Nat. Assn. Prev. Consumption, London, 1913, p. 57.
[2] Ann. de l'Inst. Pasteur, 1914, **28**, 569; Compt. rend. Soc. de biol., 1914, **76**, 197.
[3] Ibid., **28**, 338.
[4] Compt. rend. Soc. de Biol., 1914, **76**, 199.
[5] Lancet, 1914, **2**, 485. [6] Quarterly Jour. of Med., 1915, **8**, 339.
[7] Arch. Int. Med., 1914, **14**, 786; Proc. Soc. Exper. Biol. and Med., 1914, **12**, 48.
[8] Bull. 101, Hyg. Laborat., U. S. P. H. S., 1915.
[9] Am. Jour. Med. Sci., 1915, **150**, 781.

which react with the syphilitic serum in a manner similar to tissue-extract antigen, is correct, it does not help us in our efforts to find a specific test for active tuberculosis.

Various authors report between 40 and 95 per cent of positive results with the complement-fixation test. Some state that a positive reaction means an active tuberculous process somewhere in the body. McIntosh and Fildes state even that a small lesion may not reveal itself by this test: "The lesion must be of considerable dimensions before the reaction can detect it. A caseous bronchial gland will not give a positive reaction; indeed, the common affection of the cervical glands will usually yield a negative result. On the whole, we have come to the conclusion that a lesion in order to give positive results must be of such dimensions as to constitute 'disease' and require the intervention of the physician or surgeon. We look upon the positive reaction, therefore, as indicating 'active tuberculosis.' " On the other hand, Craig found that 65 per cent of clinically inactive cases of pulmonary tuberculosis gave positive reactions. Most writers obtained positive reactions in patients with syphilis.

This test has been given an extensive and careful trial in my wards at the Montefiore Hospital. My associate, Dr. H. R. Miller, has applied it in a very large number of cases. But I have not been impressed with its reliability as a diagnostic agent when we attempt to discriminate between tuberculous infection on the one hand and active tuberculous disease on the other. In many active cases it has been negative, while many non-tuberculous cases showed positive results. It appears to be of about the same value as the von Pirquet skin reaction. It, as a rule, discloses infection. To discover infection, the skin reaction may be applied with less trouble.

The Autourine and Autoserum Test.—The search for a biochemical test for active tuberculous disease has recently brought forward another reaction. Wildbolz[1] believes that he has discovered that when a tuberculous process is active in any part of the body the urine contains an antigen which produces circumscribed redness and infiltration when injected intradermally by the Mantoux method. He reported that urine from healthy individuals, when injected into the skin of tuberculous patients, does not produce this reaction, nor do healthy persons, or even tuberculous patients but in whom the lesions have healed, eliminate this antigen with the urine. Moreover, he found that the reaction goes hand-in-hand with the tuberculin reaction; the response is positive when the tuberculin test is positive, and the reverse. His conclusion is that the urine in tuberculous patients eliminates specific substances akin to those contained in tuberculin, and that they may be utilized for diagnostic purposes when the problem is whether the disease is active or healed.

As a rule, the morning urine is used. It is evaporated in a vacuum

[1] Cor.-Bl. f. Schweiz. Aertze, 1919, **49**, 793.

to one-tenth its volume, and after it is cooled it is passed once or twice through a paper filter impreganted with a 2 per cent solution of carbolic acid. He found that no results are obtained with unconcentrated urine, while when concentrated at a high temperature on an open water-bath the reaction is but weakly positive. Wildbolz makes three sets of inoculations at an interval of 2 to 4 cm. on the arm. Two inoculations are made with the concentrated urine, two with 1 to 1000 tuberculin, and two with 1 to 10,000 tuberculin. When the test turns out positive, the infiltration and redness are of the same degree at each point of inoculation. The only difference to be noted is that the tuberculin reaction persists for some time, while the urine reaction is evanescent and fades away completely within one or two days. Wildbolz,[1] and Imhof[2] found that the antigen is not only found in the urine, but also in the blood serum which has been freed from its proteins. This could be expected considering that the antigens are brought from the tuberculous lesion to the organs of excretion, the kidneys, by the blood stream. Imhof found that the autoserum reaction is, as a rule, slightly weaker than the autourine reaction, but while in the latter necrosis occurs at times at the point of inoculation, this never occurs with the autoserum reaction.

Clinical tests made by various authors have not altogether sustained Wildbolz's experiences. Some authors report that it is of value in differentiating active tuberculosis because healed and encapsulated lesions do not respond. This has been reported to be the case by Lanz,[3] Cepulic,[4] Alexander,[5] Cole B. Gibson and William E. Carroll[6] in this country, and others.

More intensive study has, however, shown that this test is of no more clinical value than the tuberculin tests. Trenkel[7] is inclined to the idea that the reaction is not of a biological nature, but purely chemical. He found that in moribund patients, in whom the tuberculin test was negative, the autourine test was positive. He also found that when a specimen of urine was partly concentrated in a vacuum, as directed by Wildbolz, and partly in an Erlenmeyer flask without a vacuum, necessitating a higher temperature for evaporation due to its being carried out at atmospheric pressure, the results obtained were the same in each case, though the high temperature in the Erlenmeyer flask has undoubtedly an effect on the thermolabile constituents of the toxins. For this reason he is inclined to attribute the reaction occurring on the skin to the salts of the urine and not to its toxin contents. Moreover, he obtained positive results with an "artificial urine." Alexander found that urine containing staphylococci also gives a posi-

[1] La Presse médicale, 1920, p. 782.
[2] Schweitz. med. Wchnschr., 1920, **50**, 1033.
[3] Schweitz. med. Wchnschr., 1920, **50**, 321.
[4] Beitr. z. Klin. d. Tuberkul., 1921, **46**, 435.
[5] Ztschr. f. Tuberkul., 1921, **33**, 321.
[6] Jour. Am. Med. Assn., 1921, **76**, 1381.
[7] Beitr. z. Klin. d. Tuberkul., 1921, **47**, 219.

tive reaction. Weisz[1] applying fractional analysis to the urine, failed to discover any specific antigens corresponding to those found in tuberculin, and he concludes that this method is of no value in differentiating active from latent tuberculous lesions. Gramen,[2] Eliasberg and Schiff,[3] Reinecke,[4] and many others, have found that clinically the test does not work out. Many non-tuberculous persons give positive reactions, and the reverse. Finally, Farrago and Randt[5] found that patients react to urine voided by persons who showed not the slightest evidences of clinical tuberculosis, such as physicians, nurses, orderlies, and hospital patients suffering from slight ailments. Their conclusion is that the reaction is of traumatic and chemical origin and not specific, and for these reasons cannot be considered of diagnostic value.

Other Special Tests.—Most of the other special diagnostic tests which have been brought forward from time to time have been found wanting in reliability; their limitations preclude their general adoption. Arneth's blood-picture has never been considered of diagnostic value and was only urged as of prognostic significance (see p. 244). Wright's opsonic-index method has been given a very extensive trial, especially in English-speaking countries, but has been found unreliable. The results are very conflicting and the method is altogether unsuitable for general adoption.

[1] Med. Klinik, 1921, **17**, 950.
[2] Hygeia, 1920, **82**, 674.
[3] Monatschr., f. Kinderheilkunde, 1920, **19**, p. 1.
[4] München. med. Wchnschr., 1920, **67**, 1202.
[5] Deutsch. med. Wchnschr., 1921, **47**, 919.

CHAPTER XX.

CHRONIC PHTHISIS, ADVANCED STAGE.

Duration of the Incipient Stage.—Incipient phthisis is also called "early" phthisis, and thus confusion is engendered in the minds of the laity, as well as of physicians, who assume that a case is incipient only for a certain time and then progresses to the second or third stage, unless properly treated. This is wrong. *There are cases which are "advanced" soon after the active symptoms manifest themselves, while others, though remaining active for years, never pass beyond the stage of incipiency.* Indeed, we meet with numerous patients who have been tuberculous for many years, and have been admitted to sanatoriums several times as "early" cases.

The sagacious clinician Laennec, stated nearly one hundred years ago that it appeared to him that hardly any consumptive succumbs to the first attack of the disease, and that in the vast majority of cases the first attack is erroneously diagnosticated as a mild respiratory trouble. The disease then remains latent for a longer or shorter time to break out again, perhaps with greater severity. Many years of research along scientific lines have confirmed Laennec's observation. A large number of cases never become "advanced" in the sense we use this term. Others show greater activity, and the process in the lungs proceeds from infiltration to caseation, softening and excavation within six months or a year. A large proportion of active cases remain quiescent for one or two years, and then suddenly take a turn for the worse and the patient sinks, succumbing to exhaustion, or to some complication.

On the whole it may be stated that *in the clinical sense, "incipiency" does not necessarily imply earliness of the process.* As used by physicians and understood by patients, it means a limited and circumscribed lesion which is not manifesting a tendency to acute progression, but either remains quiescent or leans to cicatrization of the lesion. In this stage the patient may remain for many years and no average duration can be assigned. It can only be estimated in the individual patient, depending as it does on so many different and complex factors which have been discussed elsewhere in this book.

Course of Incipient Phthisis.—In a large proportion of cases phthisis does not pass beyond the so-called stage of incipiency. The patient coughs, expectorates, has fever, hemoptysis, etc., for several weeks or months, and, after taking a rest in the country, spending a few months in a sanatorium, or even while continuing at his occupation, he slowly

recuperates and recovers, never to be troubled again with pulmonary symptoms. In most of these cases there are left remnants of the pulmonary lesion in an apex, manifesting themselves in impaired resonance, prolonged expiration and sibilation. This conforms to the abortive type of tuberculosis which will be discussed later on (Chapter XXI).

But in many cases the disease progresses steadily, especially when no proper treatment has been instituted, and occasionally irrespective of the treatment. In a small proportion of cases the progress is rather rapid, and within one or two months after the first symptoms have appeared, the patient is a confirmed consumptive; while in others the course is slower, the patient keeps on coughing, expectorating, losing flesh and strength for several months or years, when a change takes place and he is apparently improved or cured, or he succumbs to the disease.

In the vast majority the progress of the disease is marked by distinct remissions, during which the patient feels comparatively well, is able to pursue his vocation, and he, as well as his physician, is under the impression that a permanent cure has been attained, to be undeceived, now and then, by the appearance of an acute exacerbation of the disease during which the patient is laid up for several days or weeks, or by a pulmonary hemorrhage, which may or may not be copious; an attack of pleurisy, with or without effusion, etc.

There is another class of cases in which the focus in the lung remains quiescent, but does not cicatrize for many years. Physical examination of the chest shows distinct signs of an active pulmonary lesion and an examination of the sputum may even disclose tubercle bacilli, but the symptomatology and course are benign—the cough is mild, there is no fever, no nightsweats, no emaciation, and the patient is capable of working at his vocation for years. These may be considered "carriers." Though harboring tubercle bacilli in their lungs, and disseminating them with the sputum, they are themselves fairly healthy.

Oscillating Course of Chronic Phthisis.—*A continuous course from bad to worse till the patient dies, or with improvement till he recovers, is uncommon in chronic phthisis.* It is characteristic of either the abortive form of phthisis, on the one hand, or of acute galloping phthisis, on the other. But *the usual case of chronic phthisis pursues a discontinuous, paroxysmal, I may say a capricious course, marked by periods of acute or subacute exacerbations of the symptoms, and periods of remissions during which the patient is more or less free from the troublesome symptoms,* or he may even feel comparatively well, working efficiently, especially if he is engaged in some intellectual pursuit. I have seen many who have worked at hard manual labor for months until an acute exacerbation laid them up for several weeks, but they sooner or later recuperated and went to work again, until another acute exacerbation interfered.

These acute exacerbations during the course of chronic phthisis

usually have distinct pathological substrata. In active phthisis the affected part of the lung caseates, softens and is finally eliminated by cough and expectoration, leaving a fistula to drain the excavation, which is surrounded by a fibrous capsule that inhibits or prevents absorption of toxic matter. The patient may feel comparatively well so long as the cavity in the lung is well drained. But now and then the fistula is obstructed, or a new area becomes involved by contiguity or metastasis, and again acute symptoms of constitutional toxemia make their appearance. This acute exacerbation keeps on for some time till either the fistula opens again, or the newly involved area has softened, the products of tissue disintegration are eliminated, and the patient feels well again, though he is by no means cured.

This undulating course of phthisis can be clearly observed by studying the temperature, expectoration, emaciation, etc., of the patients, as was done by Bezançon,[1] Serbonnes,[2] and others. It may keep on for many years. In most cases one of two things finally occurs—either the infiltrated or excavated area in the lung cicatrizes, or becomes encapsulated and shrinks and the disease is arrested; or, during one of these exacerbations, the pulmonary involvement becomes too extensive, and can no more become quiescent and, with or without some complication, the patient succumbs.

We may say that during the long course of chronic phthisis there is an intense struggle between the bacilli and the resistance of the host. We have seen that everybody possesses more or less resistance; else every infection would speedily prove fatal. In this struggle the bacilli gain the upper hand for a time and cause an acute exacerbation, but the innate resistance is again called upon and usually responds, the result being a truce, until the bacilli again catch the organism napping. The final outcome depends on many and complex factors which are discussed elsewhere.

Symptoms.—The cough, which may have been mild during the incipient stage, gradually becomes more and more annoying and productive. It may be painful, paroxysmal and exhausting, and end in vomiting, especially after the evening meal. But with the advance of the process the cough is ameliorated in most cases; while it does not cease altogether, it becomes "looser"; the sputum is brought up without great effort. During acute exacerbations it is usually aggravated, often painful, due to complicating dry or moist pleurisy, etc. In some cases the cough is mild throughout the course of the disease, while in others it constitutes the main complaint of the patient. In fatal cases it may be absent during the last few days of life, when the reflexes are abolished, or, because of severe emaciation and muscular atrophy, the patient has not enough strength for the efforts at coughing.

The mucoid sputum of the incipient stage becomes more and more mucopurulent with the advance of the disease, and almost invariably

[1] Paris médical, 1911, p. 133.
[2] Les Poussces évolutives de la tuberculose pulmonaire chronique, Paris, 1910.

contains tubercle bacilli. Exceptionally, none are found in a case that keeps on progressing, even to a fatal issue. But this is exceedingly rare. Elastic fibers are, however, found in practically all cases in which the disease has passed incipiency, owing to the destruction of lung tissue during caseation and liquefaction. Immediately before and during an acute exacerbation the amount of sputum may be diminished, but within a few days it again increases in quantity. With the disintegration of lung tissue and formation of vomicæ, the character of the sputum changes; it becomes thick, nummular and sinks in the water of the receiving vessel. During hemorrhages it is sanguineous, and often without any evident hemorrhage it is tinged with blood. During quiescent periods the amount expectorated is, as a rule, diminished; it may lose its purulent character and, when a cure is established, the expectoration may cease. In fatal cases we often note that during the last few days little sputum is brought up. The patient has not sufficient strength to expel it, as has already been mentioned.

The temperature in active advanced cases is not of a characteristic type. In progressive cases it may be continuous or remittent till the end—recovery or death. Usually the curve, when studied for several months continuously, pursues an undulating or cyclic course. For several weeks it is high, no matter what type it is, rising to 101°, or even 104° F. in the afternoon, and declining several degrees in the morning, in many cases even to a subnormal degree. Slowly an improvement is noted, the temperature becomes lower and lower and we may find a period of either subfebrile or even normal temperature for a few weeks. In many cases I have noted a subnormal temperature for comparatively long periods.

But suddenly—perhaps after a chill or some indiscretion—or gradually, the temperature rises again and keeps at a high level for several days or weeks, thus marking an extension of the process to a hitherto unaffected area of the lung, or some complication.

It is noteworthy that, as a rule, during the afebrile periods the patient feels quite well and, for weeks, may consider himself cured, to be sadly disappointed during the acute exacerbations which are sure to come in most cases. Even during febrile periods many feel comparatively well and have a good or fair appetite, as was already stated. The intellect is usually clear; those engaged in intellectual pursuits may follow their vocations during the exacerbations. I have had patients who did business on a high scale under such circumstances, and writers and artists who produced their best work while the thermometer registered 103° F. The euphoria, which is characteristic of phthisis, is best observed in far-advanced cases.

Emaciation goes hand-in-hand with other constitutional symptoms, especially fever. Those who have no quiescent periods lose flesh very rapidly, and within a few months may be reduced to mere skeletons. In those in whom the disease runs an undulating course, we often note a gain in weight during afebrile periods, and if the fever is mild

during acute exacerbations and of short duration, the loss in weight may be insignificant. They may be ahead in this regard at the end of a year or two, although the process in the lungs remained stationary, or has even progressed.

Toward the end the emaciation is very pronounced and deserves the name consumption. Then it is not only the fever, cough, and expectoration that are exhausting the patient, but also the lack of nourishment owing to anorexia, diarrhea, and perhaps dysphagia when the larynx is implicated. The preservation of the body weight, which is very frequent in fibroid phthisis, is only rarely seen in chronic progressive phthisis, and, when found, it is an indication of improvement, or that the quiescent periods are of long duration.

Hemoptysis is comparatively infrequent during this period, excepting in very advanced cases with cavities, when a terminal hemorrhage may carry off the patient, and in those suffering from hemorrhagic phthisis (see p. 240) it may recur at irregular intervals. As was already stated, most of the hemorrhages at this period, even when profuse, end in recovery.

The other symptoms of chronic phthisis have already been described in detail in previous chapters.

Physical Signs.—Depending on alterations in the pulmonary parenchyma, pleura, mediastinum and chest walls, the physical signs of advanced phthisis are complex. By percussion and auscultation we may determine, with a reasonable degree of certainty, the nature of the lesion, as well as the condition of the apparently unaffected parts of the thoracic viscera. But with the progress of the disease, the changes found on physical exploration become more and more variegated and, owing to frequent overlapping of pathological changes, their complexity is so great that it is often quite difficult or impossible to determine exactly the details of these changes by physical examination. This is well illustrated by the difficulties encountered while attempting to determine the presence or absence of pleural adhesions before inducing a therapeutic pneumothorax and by the number of cavities that are missed during life and found at necropsy. Roentgenography is of immense value at this stage, but it is not infallible, as has already been shown.

Percussion.—The tuberculous infiltration usually extends in horizontal planes; metastatic deposits of tubercle at a distance from the original focus in the same or the opposite lung are only rarely found. The result is that the impairment of resonance found over one apex during the incipient stage extends mainly downward, and, in progressive cases, we soon find dulness as far as the third or fourth rib, or lower. The pitch of the note depends on the density of the infiltration, on the presence or absence of excavations, the amount of secretions in the cavity, and on the condition of the pleura. On the unaffected side a hyperresonant note may be elicited, which may be accentuated by vicarious emphysema.

Dulness is very frequently found in the interscapular spaces, which may be an expression of enlarged peribronchial glands, or infiltration of the apex of the lower lobe of the lung. In the majority of cases there is more or less retraction of the base of the lung, easily made out by tidal percussion.

With percussion we may also determine the position of the heart which in many cases is of immense diagnostic significance, as has been pointed out elsewhere by the writer.[1] *In phthisis the heart is, as a rule, dislocated toward the affected side, the reverse of conditions found in pleural effusions, pneumothorax, intrathoracic neoplasms, etc.* It is therefore important to determine the position of the heart in cases showing intense dulness of the lower parts of the chest on one side when the problem arises whether it is due to an effusion, or to thickened pleura with pulmonary retraction. Exploratory puncture, if negative, is not conclusive, but when we find the heart displaced to the opposite side, we may conclude that there is an effusion, while when it is dislocated toward the affected side, it is due to excavation and to pleural thickening. But to this there are many exceptions which are discussed elsewhere.

The routine methods of physical exploration show the location of the heart in phthisis easily and vividly; but in many cases the diagnosis is difficult and occasionally almost impossible. The side of the heart adjoining the healthy lung is easily made out by percussion, but the cardiac dulness at the side adjoining the affected lung merges with the dulness of the infiltrated and consolidated lung tissue or thickened pleura, and it is difficult to separate by any method of percussion. The fluoroscope and the roentgenographic plate also fail at times to show a definite outline of the borders between the heart and the lung. Indeed, I have found at times that orthodiagraphy was of no avail.

Dextrocardia is not rare in extensive right-sided lesions. It is to be differentiated from complete transposition of the viscera by the location of the liver, spleen, etc.

Auscultation.—Auscultation in advanced phthisis is of even greater diagnostic significance than percussion and roentgenography, because it shows distinctly the progress of the process in the lungs, especially its activity. The diagnosis of a healed lesion can only be made by a study of the constitutional symptoms, and a careful consideration of the auscultatory phenomena elicited over the chest.

The breath sounds which, during the incipient stage, may have been somewhat altered, rough, cog-wheel or feeble, now become more and more bronchial or tubular in character. Excepting in very acute cases, which do not concern us here, bronchial breathing does not appear suddenly in chronic phthisis. Following a progressive case we may observe that the cog-wheel breathing changes by degrees; first the expiratory murmur becomes prolonged, then the sounds assume a

[1] Arch. Int. Med., 1914, **13**, 656.

bronchovesicular character, indicating that the breath sounds are mixed, the vesicular coming from the healthy lung and the bronchial from the disseminated infiltrated patches. When these patches conglomerate, and the part of the lung consolidates into an extensive airless area, thus acting as a good conductor of the laryngo-tracheal murmur to the surface, we get bronchial breathing. With the onset of softening the products of tissue disintegration are expelled, leaving an excavation and we often, though not invariably, hear cavernous or amphoric breathing, which will be discussed later on.

The advance of the lesion is characterized pathologically by softening of lung tissue, followed by liquefaction and cavity formation. These changes are best determined by auscultation and the detection of moist rales which are produced by the air current passing from the bronchi into the diseased area filled with morbid secretions and débris of disintegrated tissue. These rales are of various sizes—large, medium or small—according to the size of the bronchus, or the excavation in which they are produced. Usually they are consonating, ringing and either provoked, or intensified, by cough. Their diagnostic significance lies in their localization and persistence. They are mostly found over the supraspinous fossæ, in the upper part of the interscapular space, and especially above and below the clavicle, and with them we usually hear low-pitched, bronchial breathing. When heard unilaterally and persistently in any of these locations, they are, with but few exceptions, pathognomonic.

The onset of softening is characterized by the appearance of moist rales, usually small or of medium size. They have been called by the French *râles de friture* because they simulate the sounds heard when frying something. But we must guard against overestimating the extent of the disease by wide distribution of rales. With concomitant bronchitis they may be distributed all over the chest, or all over one hemothorax, while the tuberculous lesion is rather limited. After pulmonary hemorrhages rales may be heard far away from the tuberculous area, and we must be guarded in concluding that it is an indication of widespread extension of the tuberculous lesion. The thermometer is a better guide under such circumstances. After an attack of influenza there may remain a large number of rales which disappear in time.

On the whole, it can be stated that the activity of the tuberculous process may be gauged by the number, character, and distribution of moist rales audible over the chest. The larger their number, the larger their consonance, when localized over a limited area, the more active the process, while absence of rales indicates an arrest in the progress of the disease. All this is true when, in addition to these adventitious sounds, there are also constitutional symptoms, especially fever. In the absence of toxic symptoms, rales may be heard in chests with quiescent, or even arrested tuberculous disease.

Sibilation is quite frequently heard in cases of advanced phthisis

and it may be caused by various conditions. In the interscapular spaces, and near the two sides of the sternum, whistling sounds are an indication of tracheo-bronchial adenopathy with pressure on the bronchi. In some cases, we hear sonorous rales all over the chest, or unilaterally, in cases complicating bronchitis or emphysema; over areas of localized vicarious emphysema, sibilation is also heard at times. For a long time, or permanently after a lesion has healed, there may remain sibilation, "cicatricial rales."

Friction sounds are very frequently heard. Their significance is discussed in connection with pleurisy.

Cavities.—This stage is characterized by the formation of pulmonary excavations. The constitutional symptoms accompanying the formation of cavities depend on the acuteness of the process. So long as the excavation is surrounded by infiltrated and caseated lung tissue, the symptoms are acute—high fever of a continuous, or remittent type, profuse nightsweats, severe cough with abundant expectoration, rapidly progressing emaciation, etc. But in most cases the process is not so acute. The excavation is surrounded by a fibrous shell which limits its progress, and prevents absorption of the toxic products to a great extent, so that the patient may feel quite well despite the formation of more or less extensive excavations in his lungs. In the chronic cases that do not succumb, but do not heal either, the cavity may keep on secreting mucopurulent matter which is promptly removed through the fistulous tract that leads to a bronchus.

It is in these chronic cavitary cases that we meet the undulating clinical picture of phthisis described above. Whenever the fistulous tract leading from the cavity is obstructed, the amount of expectoration is diminished and fever, nightsweats, etc., result, till the plug in the bronchus is dislodged, when expectoration begins to drain the cavity and the patient again feels comparatively well.

Diagnosis of Cavity in the Lung.—If we should accept the signs given in text-books as infallible criteria, the diagnosis of cavities is very simple. But those who often make autopsies and have opportunities to verify their findings are frequently amazed at the large number of cavities found *intra vitam*, but missing at the autopsy, and the reverse.

In order that a cavity should be discerned by physical exploration, or even by roentgenography, it must attain the size of at least four centimeters in diameter; it must be superficially located, filled with more air than secretions and communicate with a bronchus. In the apex cavities are often missed because the thick, indurated pleura screens all signs. Some even maintain that they must have smooth walls if we are to elicit by auscultation and percussion the signs which are characteristic of excavations. In fact, many authors who have studied the physical signs of vomicæ, verifying their findings at necropsies, found that many excavations are overlooked, while others that are diagnosed are not found at the autopsy. For this reason some believe that the presence of elastic tissue in the sputum is the best sign of pulmonary excavation.

Inspection and palpation are of little value. The muscular atrophy noted over deep excavations above and below the clavicle may be seen in pulmonary retraction without excavation. Over superficial cavities, extreme atrophy of muscles and integuments of the area overlying the excavations is very frequent. This atrophy leaves the chest wall over a circumscribed area very thin and, combined with pleural adhesions and retraction, may cause a cup-shaped depression localized over the site of the cavity, which is pulled in during inspiration. But this is comparatively uncommon, probably because many cavities are situated deeply within the lung.

Percussion over a cavity gives a dull note, and only over large excavations, superficially located in the infraclavicular region of emaciated patients, and filled mostly with air, may be obtained a hyperresonant or tympanitic note. At most, we usually find dulness with tympanitic overnote. But to indicate excavation, even this must be strictly localized and circumscribed. The resonance may change within a single day from tympany to dulness when it fills up with secretions. From the roentgenograms on Plate XVII it will be seen that percussion of this chest would have elicited a dull or flat note one day, while the next, it would have been hyperresonant or tympanitic.

On the whole, *cavity tympany* depends on many factors. In young persons, with elastic and resilient chest walls, it is more often present over small excavations than in the aged, whose chests are usually rigid and unyielding, and even large excavations may not be tympanitic. The more superficial the location, the more pronounced the tympany, while deeply lying cavities are screened by air containing lung tissue and tympany is altogether absent. It is thus evident that *tympany is not a constant sign of cavitation*, but when localized, circumscribed and pronounced it speaks for a cavity of large size with greatly relaxed walls; and conversely, we find high tympany over tight walls of small cavities. It may best be perceived, as Flint showed long ago, when the ear is close to the patient's mouth, or when the bell of the stethoscope is held in this position. Cracked-pot resonance is also best perceived in this manner.

The most common site of tympany due to cavitation is above the fourth rib anteriorly, and on rare occasions we find it in the axillary line beneath the fifth rib, especially in the left side, while posteriorly it is exceedingly rare because of the large muscles which interfere with percussion. I have met with cavities that were tympanitic over three-fourths of the chest wall, indicating excavation of almost an entire lung. But this is rare because in such cases the mediastinum is pulled over and produces dulness.

Occasionally the tone changes known as Wintrich's, Friedreich's and Gerhardt's phenomena are of assistance in the diagnosis of vomicæ, but not so frequently as some text-books would lead us to believe.

Wintrich's phenomenon, obtained by percussion while the patient opens and closes his mouth, the note being tympanitic when it is

open, and of lower and deeper pitch when closed, is a good indication of a cavity communicating with a bronchus and is more distinct the greater the diameter of the bronchus. It may be obtainable only in certain positions of the body (*interrupted Wintrich*), which is clearly due to the presence of fluid secretions within the cavity which obstruct the opening of the bronchus. It is also met with in some cases of bronchiectatic excavations, but this is to be distinguished by the location of the cavity—anteriorly and above in tuberculosis, and posteriorly and below in bronchiectasis. It may also be found in pneumothorax, but the concomitant symptoms and signs clear up the diagnosis, excepting in the localized and latent forms, which can only be recognized with the *x*-rays.

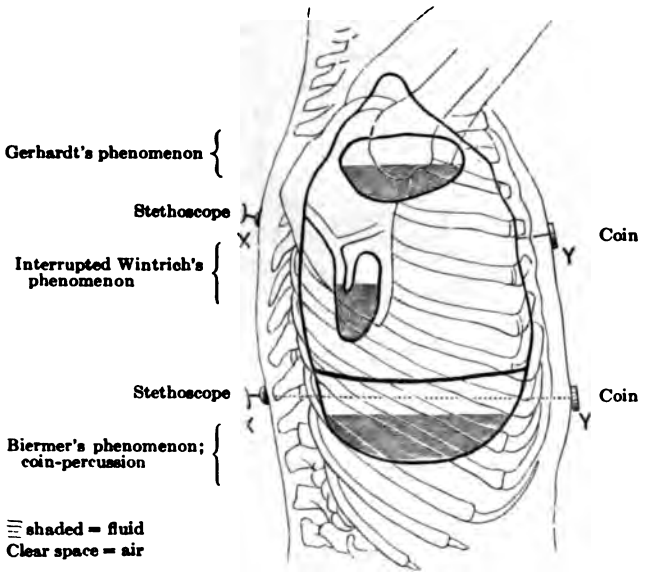


FIG. 85.—Illustrating Gerhardt's and Biermer's phenomena, interrupted Wintrich's phenomenon and coin-percussion. (Musser.)

William's tracheal tone, observed while percussing the consolidated apex which conducts the tracheal tympany, is at times mistaken for Wintrich's phenomenon. It is usually found in cases of contraction or consolidation of lung tissue, or its compression in pleuritic exudates, when percussion above and below the clavicle sets up vibrations in the main bronchus and the trachea.

Friedreich's phenomenon consists in high-pitched tympany over the site of excavations when the patient holds his breath during full inspiration, diminishing during extreme and held expiration. This is not so reliable as Wintrich's sign because it is at times obtained over healthy lungs.

In *Gerhardt's phenomenon* the note is higher and more tympanitic when the patient is sitting or standing than when he is reclining, and is said to be characteristic of an oval-shaped cavity filled partly with fluid and partly with air, the fluid gravitating according to the position of the patient. Small cavities, superficially located, occasionally show this sign and when the excavation is centrally located, it must attain considerable dimensions to be thus characterized. As Sahli points out, Gerhardt's phenomenon is rare, and slight differences in the percussion note with changes in position may be within physiological limits due simply to alteration in the tension of the thoracic walls without any cavity within the chest.

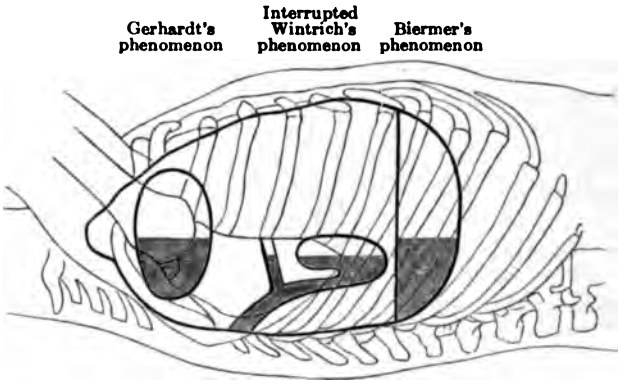


FIG. 86.—Illustrating Gerhardt's and Biermer's phenomena and interrupted Wintrich's phenomenon. (Musser.)

In hydropneumothorax we often observe *Biermer's phenomenon*, which is produced in the same manner as Gerhardt's in pulmonary cavities (see Figs. 85 and 86). This shifting of fluid in pneumothorax is discussed more fully in another portion of this book (see Chapter XXVII).

Cracked-pot resonance, first described by Laennec, is occasionally obtained over cavities. Some precautions are necessary in order to elicit this sign. The patient should keep his mouth wide open, the plexímeter finger placed over the second or third intercostal space anteriorly, and with the percussion finger a strong blow is delivered without rebound, at the end of expiration. It is apparently a stenotic murmur at the opening of the cavity into a bronchus when the air is suddenly expelled through a narrow, slit-like opening. Plesch[1] says that for the production of the cracked-pot resonance the cavity must be surrounded by elastic tissue which helps in producing a double motion of the air—a sudden expulsion from and quickly sucking it back into the cavity. It is imperative that the two pleural sheets overlying the cavity should be adherent and the thorax should be

[1] Deutsch. med. Wchnschr., 1917, **43**, 175.

resilient and elastic. It may, however, be met with in many other conditions, as in a crying child, and in adults with relaxed lungs, also in emaciated persons with resilient chest walls, and in cases of small emphysematous islands surrounded by consolidated lung tissue which are not uncommon in chronic phthisis. Of the many cavities that I have seen, cracked-pot resonance was present in but a small proportion. When obtained in connection with some of the other signs it is of significance.

Cavernous and Amphoric Breathing.—Auscultation may be altogether negative over deeply lying vomicæ, or such as are completely closed by a plug in the communicating bronchus. Cavernous breathing is often heard; it resembles the sound produced while blowing into an inclosed hollow space. It is caused by the overtones developed in the cavity by reflection from the walls. Over cavities having smooth walls communicating with a bronchus we often hear amphoric breathing—a murmur with high overtones lacking deep basal tones, resembling the sound produced by blowing across the opening of a narrow-mouthed vase. Cavernous and amphoric breathing have a certain diagnostic significance. They indicate pulmonary excavation, bronchiectasis, or pneumothorax. Formerly it was thought that pneumothorax shows amphoric breathing only when it is freely communicating with a bronchus. But now we often find it over artificial pneumothorax, and it is then due to reverberation of the bronchial sounds from the smooth pleura. Over many excavations only loud and harsh bronchial breathing is audible.

Over areas with amphoric breathing we usually elicit a dull note on percussion and, at times, cracked-pot resonance, while over areas with cavernous breathing we often get tympanitic resonance, though not always, as was already indicated. Amphoric resonance is an indication that the excavation is at least five centimeters in diameter, that its walls are smooth, round, and rigid, due to surrounding infiltration or fibrosis; that in all probability it communicates with a bronchus of not very wide caliber; and that it is not active—a fibrous capsule prevents the absorption of toxic matter from the cavity, and also the extension of the lesion, and the small amount of secretion is soon eliminated by expectoration. It is for these reasons that cavities with amphoric breathing are usually not accompanied by any adventitious sounds, excepting at times by a metallic tinkle, and this is very rare; while cavernous breathing is almost always accompanied by large or medium-sized consonating rales or gurgles. In the latter case the cavity is active, probably growing, and not surrounded by a fibrous shell. The prognostic significance is clear. The intensity of the amphoric phenomena depends on the stiffness of the wall which, in its turn, depends on a strong fibrous capsule, or an infiltration and caseation of the surrounding lung tissue. In the former case it will not enlarge and may even shrink, while in the latter case the excavation may extend and usually does.

Metamorphosed Breathing.—Over the sites of cavities, mainly over the upper lobes, we sometimes hear the inspiratory murmur begin as a harsh or bronchial murmur, but during its course suddenly softens and changes in tone, finally ending with an amphoric sound. At times, both inspiration and expiration are thus affected. Laennec spoke of it long ago as a *souffle voilé*, beginning as vesicular and ending as bronchial or amphoric. It seems that it is due to the breathing of a cavity. The air enters into a relaxed excavation and the murmur is modified, while its walls are being distended or inflated. According to Flesch it occurs only when there are slight pleural adhesions over the cavity, and when there are small islands of airless tissue or cavities scattered in an air containing, but relaxed parenchyma. On the other hand, Riess[1] found that it is audible over cavities into which two to four bronchi enter. During inspiration at first one bronchus is open while the others happen to be plugged; but soon another one or more of the entering bronchi open and introduce another type of sounds. It is one of the best signs of an excavation, but it is only rarely met with.

The India-rubber Ball Sound.—A very reliable sign of a cavity is the post-tussive suction sound heard immediately after an explosive cough. The patient is made to cough vigorously once or twice while the chest is auscultated over the site of the excavation. A hissing sound, as if another inspiration had taken place immediately after the patient ceases coughing, is heard. At times this post-tussive suction is felt with the palpating hand laid over the chest wall. It appears that this phenomenon is heard only in patients with cavities with yielding walls, which become more or less compressed during cough, and reëxpand immediately after the cough, sucking in air, thus producing a hissing sound plainly audible through the stethoscope. It is this sound that suggested the analogy with the india-rubber ball. It is usually heard over pulmonary cavities, but Bergmark reports that it may also be heard over pleural adhesions.

Adventitious Sounds Heard over Cavities.—Over excavations, large moist, bubbling, consonating rales—called in text-books metallic or cavernous rales—are often heard. They are caused by the air stream passing through the collection of fluid in the excavation. The size, pitch, timber, and duration of these rales depend on the size of the vomicæ in which they originate, as well as the condition of its walls—whether they are smooth or ragged, rigid or relaxed, etc. On the other hand, over old cavities there may be audible amphoric breathing of an exquisite type, metallic breathing without any rales at all, because the fibrous walls do not secrete any more. These are cases that are doing well for years in spite of extensive excavations. In many arrested cavities there remain creaky sounds, especially when the plural sheets are not at all adherent, or when the adhesions are but partial, so that there are differences in the elasticity of various parts of the affected area of the lung.

[1] Deutsch. med. Wchnschr., 1917, 43, 392.

In many cases the number of rales in excavations and their intensity are so great that they obscure all the breath sounds.

The metallic tinkle is only rarely heard over pulmonary cavities.

Pectoriloquy is met with over pulmonary cavities, but it is not pathognomonic of this condition. In many cases we hear the voice as if it is directly spoken into the ear with abnormal clearness. It merely indicates that the conditions for conduction are unusually good, which may be true of excavations, but are also met with in pneumothorax, and even in consolidated lung tissue through which a bronchus passes.

The same is true of whispered pectoriloquy. But the transmission of the whispered voice with a metallic or amphoric echo, which Kuthy calls "amphorophony," is a sure indication of a smooth-walled cavity filled with air, either pulmonary or pleural, *i. e.*, a tuberculous excavation or a localized pneumothorax. The differential diagnosis between these two conditions can, at times, be made out by the *x*-rays, but I have met with cases in which roentgenography was not decisive. Some cavities can be made out by auscultation with much less trouble and greater reliability than by other diagnostic methods. Amphorophony is, however, only audible over old and larger cavities which are stationary, while over acutely progressive and extending vomicæ it is only rarely heard. In many cases of localized pneumothorax I have found distinct whispered pectoriloquy in the axilla, which is exceedingly rare in cavity. This is a sign of great value in attempts at differentiation between these two conditions.

Basal Cavities.—The vast majority of tuberculous cavities are formed in the upper lobes of the lungs, except in the terminal stages, when the resistance is very low, excavations then extending into the lower lobes of the lungs.

They are very difficult of diagnosis. We may find signs of excavations at the base which are really "phantom caverns," as William Ewart[1] called them. The amphoric sounds of an excavation in the upper lobe are transmitted to the base by some transient or permanent consolidation. Echo may also be responsible for cavernous sounds at the base when the original excavation is situated in the opposite side of the chest and not in immediate contact with the spinal column.

Basal cavities were rare in phthisical persons, but of late more of them are encountered. It seems that in many cases, even when occurring in tuberculous patients, they are of influenzal origin. Many tuberculous patients who were attacked by epidemic influenza and bronchopneumonia remained with interstitial pneumonic lesions in the lower lobes of the lung, especially the left lung. In these patients the tuberculous process in the upper lobe pursues its course almost unaffected by the bronchiectatic lesion in the lower lobe; but they cough and expectorate much more severely than those who are free

[1] Goulstonian Lectures, British Med. Jour., 1882.

from this complication. The number of this sort of cases has very much increased during recent years.

It is important to mention that the prognosis is more unfavorable in tuberculous basal cavities than in those located in the upper lobes, undoubtedly because they do not empty themselves with ease. Considering a pulmonary cavity as an abscess, we understand that when it does not drain the result must be disastrous; the abundant secretions fill it up, and cough is not very effective in removing them. In the terminal stages of phthisis with lesions in the upper lobe, excavations sometimes form at the base, as we find them at necropsy, and kill the patient who may have been getting along very well before their occurrence. In fact, if in the course of chronic phthisis signs of excavation appear in the lower half of the chest, the prognosis is very gloomy.

Visceral Displacements.—The displacements of the mediastinal organs have already been referred to (p. 401). The heart is in most cases of advanced phthisis displaced toward the affected side of the chest, and in right-sided lesions we at times meet with complete dextrocardia. But in many cases there are also to be noted displacements of the trachea and larynx, first described by E. Ruedinger.[1] Gerald B. Webb, A.M. Forster, and B. G. Gilbert[2] described in detail the tracheal position in phthisis and suggested an easy method of detecting it: By placing the hand behind the neck while the thumb anteriorly reaches out to the trachea and rolls it, we can in most cases determine its position. It appears that in most cases of early phthisis the trachea is displaced toward the affected side. Webb found in 100 cases of pulmonary tuberculosis of all ages the recognition of the side especially affected proved correct in 69, doubtful in 19, and incorrect in 12 cases. It is due to pleural adhesions, together with fibrosis in the lung or pulmonary retraction pulling the trachea along.

This deviated trachea is occasionally a source of error in diagnosis. When it is displaced to the margin of the sternum, we hear loud tracheal or even "cavernous" breath sounds both anteriorly and posteriorly, and thus diagnose a cavity which does not exist. Especially is this error of great moment when the trachea is displaced to the opposite unaffected side after the induction of a pneumothorax, and we may think that there is a cavity in the untreated lung. But a little care will usually clear up the case, especially when the possibility of displacement of the trachea is borne in mind. Webb says that movement of the trachea to the side of the healthier lung following the application of pneumothorax foretells a successful application of this procedure. In my experience this is not invariably the case.

In many cases there is also upward displacement of the stomach and liver after pulmonary retraction.

Duration of the Disease.—The duration of chronic phthisis is variable. Some patients get well, or succumb, within one year, while in

[1] Beitr. z. Klin. d. Tuberkulose, 1910, **17**, 151.
[2] Jour. Am. Med. Assn., 1915, **65**, 1017.

most the sluggish course continues intermittently for many years, during which period the patients consider themselves cured, and suffer from "relapses" several times. They constitute the bulk of the class of patients who are admitted to sanatoriums and hospitals for consumptives several times. The reason is clear when we bear in mind the oscillating course of the disease—during acute or subacute exacerbations they seek relief in institutions, while during remissions, when the process is quiescent, they believe that they have been cured, or the disease has been arrested.

Basing their estimates on heterogeneous material, different authors have estimated the average life of the consumptive as at from one to ten years. Leudet[1] found that of hospital patients 90.7 per cent die within five years of the onset of the first symptoms; 9.3 per cent during the sixth to the nineteenth year. He also found that among the more prosperous patients only 77.2 per cent die within the first five years, and 22.8 per cent between the sixth and the nineteenth years. Brown and Pope,[2] studying statistically the outlook of patients discharged from the Adirondack Cottage Sanitarium, found that of those discharged "apparently cured" at the end of five years, 94 per cent of the expected were alive; at the end of ten years, 86 per cent. In those "arrested" the proportions for the corresponding years were 63, 49 and 46 per cent; and for those "active," 25, 15 and 10 per cent. It is thus clear that "an arrested" or even an "active" case is not necessarily doomed. There are always good chances to live for long years.

The striking disparity in these two sets of statistics is due to the difference in the material. Leudet studied only fatal hospital cases, without including any of those who survived twenty years, while Brown and Pope studied cases discharged from a good sanatorium in which moderately well-to-do patients predominate, and among whom a fairly large proportion were affected with the abortive type of the disease.

Attempts at estimating the average duration of life of the consumptive have also met with failure because it is difficult to obtain comparable material. When only acute, progressive cases are considered, the average is a low figure, one year or even less; when abortive cases are considered—and they are mostly those which have been diagnosed exceedingly early in the disease—the average is very high. It is for this reason that the "averages" vary from one to ten years, according to different authors.

But for the individual patient, with whom the physician deals, averages do not count for much. He must be judged by the clinical manifestations. It may be stated that those who have long periods of quiescence live long; many practically their natural lives. They

[1] Quoted from Kuthy and Wolff-Eisner, Prognosenstellung d. Tuberkulose, Berlin, 1914, p. 56.

[2] Am. Med., 1904, **8**, 879; Ztschr. f. Tuberkulose, 1908, **12**, 205.

may be "cured" several times when they suffer from acute or subacute exacerbations, but they recuperate every time and live on, often with quite some efficiency. On the other hand, in the case of those in whom acute or subacute exacerbations are frequent, and each is of long duration, a fatal issue is inevitable sooner or later.

Modes of Death.—Death supervening during an acute exacerbation, when the process in the lungs is extending, or the toxemia is severe, or the resistance is low, may be rapid, like from pneumonia or septicemia. The patient may have done quite well, but is suddenly stricken with high fever and prostration, and he succumbs to dyspnea, cardiac failure, etc. At the autopsy acute miliary tuberculosis is found in these cases. Usually the process is slower; the high continuous or remittent fever, the profuse nightsweats, anorexia, dysphagia due to laryngeal ulceration, extreme emaciation, etc., keep on for weeks or months; the patient is gradually but surely consumed by the disease. In some, the last few weeks resemble in their symptomatology the typhoid state with marked prostration, muttering delirium, etc., which, again, is an indication of complicating miliary tuberculosis.

In others, the cachexia progresses despite the fact that the fever is low, hardly ever exceeding 101° F., and the patients finally die from asthenia, like those suffering from malignant disease. Excepting the cough, diarrhea, and weakness they do not suffer much and, because the sensorium is well retained to the end, the euphoria may be exquisite. Others consider themselves quite well despite the extreme emaciation and attempt to walk around, against the advice of their physician, and among them death due to asystole the result of toxic myocarditis—the poisoned heart of Sir James Mackenzie, may occur. Some of these unfortunates are occasionally found dead in bed in the morning. But in such cases it may not have been syncope, but a heavy dose of some opiate which abolished the reflexes, prevented cough and expectoration, and they were drowned by their own secretions. Other causes of sudden death during the night are sudden onset of pneumothorax, copious hemorrhage, etc., killing before aid can be summoned.

Complications of the disease are often responsible for a fatal issue. Among the most important are pulmonary hemorrhage and pneumothorax. While 98 per cent of patients who suffer from more or less bleeding survive the accident, 2 per cent succumb to it. The patient may feel comparatively well, and in fact consider himself on the way of recovery, or even cured, when suddenly brisk and profuse hemorrhage occurs and kills him. Emaciated patients may die as a result of suffocation with their own blood, being powerless to expel it from the chest.

Pneumothorax is the cause of death in about one of 150 fatal cases of phthisis. This may kill the patient within one or two days, the cause of death being asphyxia, or within a few weeks or months through complicating pyothorax.

Complicating laryngeal tuberculosis is responsible for the death of many patients through dysphagia, dyspnea, edema of the glottis, etc.

Between 5 and 10 per cent of deaths from phthisis are preceded by cerebral symptoms. Most of these are due to tuberculous meningitis, but some are also caused by uremia, as was already stated.

Premonitory Signs of Death.—In chronic phthisis with tendencies to a fatal issue, it is often very difficult to prognosticate the time when the end will come. Indeed, the more extensive the experience of a physician with this disease, the more guarded he becomes in foretelling the day of death. Such statements as "he cannot survive three days," or "he will surely die within a week," etc., should be avoided. Some patients keep on living for weeks or months under conditions which are puzzling, to say the least.

There are symptoms and signs which may, however, be considered precursors of death in phthisis. Of these we may mention: Dysphagia, due to laryngeal ulceration, when not quickly relieved by treatment, is a sure indication that the patient will not survive very long. The same is true of profuse diarrhea which cannot be controlled by treatment. The emaciation is extreme, and the end comes rapidly. But I have seen cases with profuse diarrhea lasting for months, in spite of the fact that they hardly assimilated any nourishment. The reason is clear when we consider that the emaciated victim of phthisis lies quietly, hardly moving a limb, or expending any energy, so that the least fuel is sufficient to keep the spark of life aglow.

Edema of the extremities very often appears shortly before death. It is usually due to cardiac weakness or nephritis, thrombosis or thrombophlebitis. It may be unilateral, but usually both lower extremities are affected. The swelling may be enormous in extreme cases, while in most it is but moderate and tender on pressure. When this edema of the lower extremities is combined with cyanosis and dyspnea, a fatal issue may be expected within a month. Thrombosis of the femoral, jugular, subclavian, or other veins is one of the surest premonitory signs of death. A sudden elevation of the temperature, accompanied by severe dyspnea, cyanosis, the typhoid state, muttering delirium, etc., is an indication of complicating acute miliary tuberculosis from which recovery is not to be expected.

Aphthous stomatitis commonly portends death. In some cases treatment may improve the condition in the mouth, but within a few weeks the powers of life wane and death supervenes. Another sign which justifies information to relatives that the end is near is a red, spongy condition of the free edge of the gums.

CHAPTER XXI.

ABORTIVE TUBERCULOSIS.

Natural Resistance Against Phthisis.—As was already shown, infection with tubercle bacilli is harmless to the vast majority of civilized people; the lesion cicatrizes more or less quickly without producing distinct clinical symptoms. During childhood, when most infections occur, the morbidity and mortality from this disease are insignificant.

We cannot recognize these mild or abortive infections clinically, except by the tuberculin test; they probably pass as slight or severe "colds," grippe, bronchitis, etc. Nor do we know whether they are due to the inoculation by strains of bacilli of low virulence, considering the marked difference in virulence displayed by various strains of tubercle bacilli. The suggestion that they may be due to infection with bovine bacilli appears to have much in its favor, but this also has not been proved.

Clinical evidence seems to point in another direction as to why some tuberculous lesions are abortive. As will be shown later on (see p. 607) individuals with normal or excessive action of the thyroid gland only rarely suffer from acute and progressive phthisis; likewise those in whom the lymphoid apparatus is hypertrophied, especially of the "lymphatic diathesis," only rarely succumb to tuberculous disease. It is in fact rare to meet phthisical patients with enlarged lymphoid tissues in the throat. When tuberculous lesions are discovered in these individuals, they are either quiescent, or altogether sclerosed, and have hardly any symptoms. In his study of such lesions, Eugene L. Opie[1] found that healed and in great part calcified lesions in persons who died from non-tuberculous disease are almost invariably accompanied by hypertrophy of the lymphatic glands in which tuberculous lesions tend to heal even when extensive. In other words, individuals with properly active lymphoid glands are likely to suffer from abortive tuberculosis when infected with tubercle bacilli. This subject deserves further investigation.

We meet at times cases of abortive tuberculosis, *i. e., patients in whom the disease, instead of pursuing the usual clinical course to its termination in death or recovery after several months' or years' illness, is aborted within a few weeks or months of indisposition.* In other words, just as we at times meet with cases of abortive pneumonia, typhoid, scarlet fever, etc., so is there a form of pulmonary tuberculosis which is of relatively short duration and invariably terminates in recovery.

[1] Am. Rev. Tubercul., 1920, **4**, 629.

In these cases the lesion is apparently circumscribed, of little activity, often altogether latent and quickly cicatrizes, and, when the patient dies from any other cause, it is found at the autopsy in the shape of more or less extensive scars located at the extreme apex, pleural adhesions, or even isolated fibrous or calcareous nodules which hardly caused any inconvenience to their owners during life.

In the older works on phthisis, this form of tuberculosis is not mentioned at all. In former days only advanced phthisis was recognized. But in recent years, since Bard[1] described the pathology and symptomatology of *tuberculose abortive*, many others have mentioned it more or less extensively. In the second edition of Cornet's[2] treatise, also in Bandelier and Röpke's book, we find it mentioned cursorily, while Piéry[3] in his book devotes an extensive chapter to it. Bezançon[4] and the present author[5] have published papers on the subject of abortive tuberculosis.

Abortive tuberculosis is responsible for a large proportion of "non-tuberculous" cases in sanatoriums—the lesion heals very quickly and it is often suspected that the patients were admitted through an error in diagnosis. Many of the patients who state that well-known physicians have considered them tuberculous at one time, but that they have none the less been healthy all along for years, have in fact been affected with the abortive type of the disease at the time the diagnosis was made. I have seen many patients who applied for admission to public sanatoriums and were passed by the admitting physicians as eligible incipient cases, but inasmuch as the institutions were overcrowded, they had to wait for weeks or months for vacant beds. When they were finally called, it was found that all the symptoms and signs of the disease had vanished. As will be shown later on (see p. 486), many of these cases are instances of apical tuberculous pleurisy. The prognosis in tuberculous pleurisy is usually better than in parenchymatous lesions. A large proportion of cases of "persistent colds," grippe, rhinopharyngitis, etc., are also abortive tuberculosis. If they were carefully studied, we would discover some physical signs in the chest substantiating this view. In fact, L. Napoleon Boston[6] reports finding tubercle bacilli in cases of acute colds, influenza, bronchitis, etc., but the patients recovered without becoming tuberculous. Many of these were in fact abortive tuberculosis.

Symptomatology of Abortive Tuberculosis.—*The symptoms and signs of abortive tuberculosis are the same as those of incipient phthisis, but they never pass beyond that stage.* In most cases it begins with the symptoms of a common "cold." After some exposure the patient begins to cough, has some fever, malaise, backache, etc., and is treated

[1] Formes cliniques de la tuberculose pulmonaire, Genève, 1901.
[2] Die Tuberkulose, Vienna, 1907, p. 690.
[3] La tuberculose pulmonaire, Paris, 1910, p. 491.
[4] Bull. Soc. hôp. de Paris, 1901, p. 933.
[5] Medical Record, 1913, **82**, 921.
[6] Interstate Med. Jour., 1914, **21**, 330.

for coryza, grippe, tonsillitis, etc. But instead of ameliorating within a few days or a week, the symptoms persist for a month or two. In many cases the onset is marked by hemoptysis. The patient, who has felt quite well, or at most has coughed for a few days, suddenly feels some irritation in the throat and coughs out some blood or blood-streaked sputum. The bleeding may last for a few hours or days and either stops abruptly, or continues for a few days in the form of streaky sputum. Many tuberculous patients give histories of one or more attacks of abortive tuberculosis years before the onset of symptoms of active phthisis. The last attack is not "aborted." Every physician has among his clientele patients who expectorated blood years ago, but have felt well all along. While in many of these the hemorrhage was of extrapulmonary origin, as was already shown, in others it was due to abortive tuberculosis.

When the thermometer is carefully and judiciously used, we find fever of a mild type; especially in the afternoon there is a rise of one or two degrees, and in the early morning there may be some subnormal temperature. In some cases that came under my observation I found the typical temperature curve of mild incipient phthisis, and there were many of the accompanying symptoms of hyperthermia—malaise, languor, pain in limbs, backache, etc. While the patient is not completely incapacitated, yet he feels tired during the afternoon, but recuperates in the evening, or feels refreshed after a night's sleep. Nightsweats are rare, but in a few I have noted that they were drenching. The appetite is usually retained and when the patient is told to eat well and plenty, he finds no difficulty in following instructions.

Cough is a constant symptom; though many state that they do not cough, careful inquiry reveals that they clear their throats in the morning. We often meet with dry, hacking cough which is an annoyance during the day, and keeps the patient awake during the night. Occasionally the cough is productive of glairy mucus, but the mucopurulent sputum of phthisis is never seen in abortive cases, unless there is some rhinopharyngitis.

Most abortive cases are of the "closed" variety of tuberculosis, but now and then we meet with one showing tubercle bacilli in the sputum. Considering that a considerable proportion of these cases have hardly any parenchymatous lesion, but are in fact instances of apical pleurisy, it is clear that tubercle bacilli in the sputum should be detected only exceptionally.

In some, we meet with hoarseness lasting intermittently for a few hours during the day, or for several days in succession.

Tachycardia is not a very frequent symptom, but we very often find instability of the pulse; the least exertion or excitement raises its rate to 90 or more per minute. The blood-pressure is usually lower than normal. With the improvement in the condition of the patient both the pulse and the blood-pressure become normal again.

Physical Signs.—The objective signs are those of incipient phthisis. Of course, when the lesion is limited and centrally located, we may not find any physical signs at all, and without hemoptysis and tubercle bacilli in the sputum, the diagnosis cannot be made. In all probability the vast majority of tuberculous infections in man are of this character. They are aborted without revealing themselves in any way. But in those in whom the conglomeration of tubercles is large enough to alter the air content in a limited area of the lung, we may find signs on percussion and auscultation.

A short note above and immediately beneath the clavicle is quite common. But this may be obscured by vicarious emphysema, hyperfunction, or relaxation, of the surrounding lung tissue which may emit a hyperresonant note. Shortening of an apex, or narrowing of Krönig's resonant areas, is more common and can be easily made out with careful percussion.

On auscultation we may hear feeble breath sounds over the site of the lesion, or rough, interrupted, cog-wheel breathing. Only the inspiratory murmur is usually altered, but I have seen cases in which the expiratory murmur was prolonged, and even bronchovesicular in character, indicating extensive infiltration, yet recovery went on speedily, showing that even a considerable focus may be aborted. This is confirmed by the large scars or encapsulated and calcified tubercles found at times while making autopsies on persons who died from causes other than tuberculosis.

Adventitious sounds are not often heard, excepting in those who have had hemoptysis and in some grippal cases, in which dry crackles, of crepitation, may be audible during inspiration and influenced by cough. In many a friction sound is audible over the supraspinous fossa, in the "alarm zone." There are instances of apical tuberculous pleurisy which is described elsewhere (see p. 486). Of course, to be of significance, these signs must be strictly localized at one apex, and constant for some time. They must also be differentiated from spurious rales, as well as from marginal sounds.

Roentgenography is of little value, as was already stated in Chapters XVII and XIX.

Diagnosis.—These are the classical symptoms and signs of incipient phthisis, and when meeting with a case we are by no means certain as to the course the disease is likely to take. In fact, many abortive cases are admitted to sanatoriums where they are speedily cured, and they contribute no small portion of the statistical success of institutional treatment.

In the progressive cases the lesion extends and the constitutional symptoms become more and more marked within a few months, while in the abortive form the mild fever, cough, nightsweats, etc., abate within a few weeks or one or two months, and the physical signs disappear, or they are superseded by sibilation, and there may permanently remain a prolonged expiratory murmur over the affected apex.

While in most cases the local impairment of resonance remains, and for this reason there are many persons in whom there are differences in this regard when the two apices are compared, I have observed that in some even this disappears, to be replaced by slight hyperresonance, due probably to hyperfunction, the result of vicarious emphysema of lung tissue around the cicatrix which was caused by the healing process.

Without observing the patient for several weeks, and without an initial pulmonary hemorrhage, or tubercle bacilli in the sputum, abortive tuberculosis cannot be diagnosticated, because there always lurks a suspicion that it may have been a non-tuberculous apical lesion. There are, however, some points which may help us in recognizing this form of tuberculosis: When a patient with a distinct apical lesion has a good appetite and normal gastric function, gaining weight and strength as soon as he begins to take care of himself, there is a likelihood that the lesion may be aborted and cured within two or three months. However, this may prove deceptive at times. Some points which have helped me are the following: A slow pulse, not much influenced by exertion or excitement, speaks for a benign process. The initial hemoptysis of chronic phthisis, as was already stated, is usually preceded by cough, weakness, nightsweats, etc., for weeks before the bleeding, while in abortive cases this is rare—the hemoptysis comes like a thunderbolt out of a clear sky, without any premonitory symptoms and without any apparent exciting cause. In progressive cases the initial hemoptysis is usually more abundant, and always followed by fever of the type described above. In abortive tuberculosis the temperature remains normal at times, but usually it is slightly elevated, 1° or 1.5° F. for a couple of weeks. Initial hemoptysis of tuberculous origin without high or moderate fever, and without tachycardia, weakness, languor, etc., points to an abortive lesion.

In the majority of cases, however, only careful observation of the course of the affection is decisive. Moreover, abortive tuberculosis is apt to recur. As was already stated, in many cases the exacerbations abate, but now and then the new attack persists and then symptoms of chronic phthisis make their appearance. At times the subsequent attack manifests itself by acute and progressive course, again confirming the observations that acute tuberculosis lesions in adults are antedated by milder attacks.

CHAPTER XXII.

FIBROID PHTHISIS.

Fibrous Hyperplasia in Phthisis.—Discussing the morbid anatomy of phthisis, we showed that while the tuberculous process is mainly one of destruction—infiltration, caseation and softening—there are reparative forces at work in almost every case, manifesting themselves principally in the formation of connective tissue which either heals the lesion through cicatrization, or at least limits its progress. In fact, it may be said that without the formation of connective tissue, every case of phthisis would be acute. The balance between the destructive and reparative processes in phthisis depends consequently on the amount of fibrosis within and about the lesion—the more intense the formation of fibrous tissue the slower the progress of the disease, and, conversely, the more caseation and necrosis, the more acute and progressive the disease.

We must distinguish between fibrosis and formation of cicatrices. When a lesion cicatrizes, the activity of the tuberculous focus is extinguished, though without any *restitutio ad integrum*, as is seen in healed tuberculous lesions of the lungs and pleura. But in fibrosis the lesion is an active, inflammatory process, though it may be only slightly progressive, yet connective tissue is being continually produced. In other words, in fibroid phthisis the destructive process is smouldering, though in abeyance, or entirely absent, and the proliferative process dominates. As Bard says, the lesions may be progressive and spreading, though they are not of a destructive character.

It must also not be confused with fibroid degeneration of the pulmonary parenchyma which at times follows acute or chronic non-tuberculous inflammatory processes of the lungs, such as the so-called interstitial pneumonia, pulmonary induration, or cirrhosis, etc. Fibroid phthisis is a specific proliferation of the lung tissue caused by tubercle bacilli.

Clinically this form of tuberculosis is characterized by an exceedingly chronic course extending over many years, finally leading, in most cases, to the development of the symptoms and course of the common form of chronic phthisis. It differs from other forms of inflammatory fibrous degenerations of the lung in that it is caused by tubercle bacilli, and that characteristic tuberculous giant cells are found microscopically in the lesions of fibroid phthisis.

Fibroid phthisis was mentioned by Bayle one hundred years ago and ever since by many others; Sir Andrew Clark[1] coined the term,

[1] Fibroid Diseases of the Lung, London, 1906.

and made a thorough study of the pathology and symptomatology of the disease. C. J. B. and C. T. Williams,[1] in their book on consumption, also give a complete description of this form of phthisis. Of the more recent writers who treat of this subject may be mentioned Bard,[2] Sokolowski,[3] and Piéry.[4] While most of the authors do not agree on the various points which characterize fibroid phthisis, yet in the main they are in agreement on its differentiation from all other forms of pulmonary tuberculous disease.

Etiology.—Fibroid phthisis is mainly encountered in persons between forty and sixty years of age and, contrary to the statements of many authors, it may occur in younger individuals. Apparently many cases are treated for chronic bronchitis, asthma, pulmonary emphysema, etc., and only after the process has lasted for many years is the character of the affection recognized; an intercurrent hemorrhage, or tubercle bacilli in the sputum, reveals the true nature of the disease. I have met with many cases in persons under thirty years of age.

It appears that syphilis is an important etiological factor; when both tuberculosis and syphilis are met with in the same individual, the process of the former is often of the fibroid type. Sergent[5] and several other French writers have indeed maintained that most fibroid cases are a manifestation of syphilis and tuberculosis. Several English authors hold the same view. Thus, J. Mitchell Bruce[6] says: "It should be noted that some cases of quiescent phthisis give a history of syphilis which may account for the disposition to fibrosis, and *pro tanto* may be a favorable element prognostically." In my experience this holds true for some cases, but not for the majority. I have seen many cases of fibroid phthisis in which specific disease was positively excluded, and at the Montefiore Hospital, where we have many of these cases, the Wassermann reaction is only rarely positive, and the other stigmata of syphilis are lacking in the majority of cases of fibroid phthisis.

English authors, notably Clark, have observed that the gouty diathesis, which is antagonistic to tuberculosis, is responsible for the fibroid form of phthisis. This is not in agreement with my experience, because among the poor in New York City gout is rather rare, while fibroid phthisis is quite common. Nor have I found any etiological relations between fibroid phthisis and alcoholism, or social and economic conditions, etc.

It appears to me that occupation is of greater etiological moment. Most of the cases I have seen were in persons working indoors, inhaling animal and vegetable dust—garment-workers, furriers, rag-pickers,

[1] Pulmonary Consumption, London, 1887.
[2] Forms cliniques de la tuberculose pulmonaire, classification et description sommaire, Genève, 1901.
[3] Klinik der Brustkrankheiten, Berlin, 1906, **2**, 410.
[4] La Tuberculose pulmonaire, Paris, 1910.
[5] Presse médicale, 1908, **16**, 657.
[6] Lancet, 1913, **1**, 591.

etc. It seems also that chronic lead poisoning is a predisposing factor, because of its frequency among plumbers, printers, and house painters. In former days it was frequently seen among chimney-sweepers, and today it is met with among those who inhale any irritative dust, as knife-grinders, coal-heavers, button-makers, etc.

Pathology.—The pathology of fibroid phthisis has been thoroughly studied by Sir Andrew Clark, who described that the affected lung is usually decreased in size; sometimes its dimensions do not exceed the size of a closed fist. In local fibrosis only the affected part of the lung may be contracted, while the rest fills up its place by compensatory emphysema. Cavities—pulmonary and bronchiectatic—are common, surrounded by dense, rigid walls. Cheesy nodules encapsulated by fibroid tissue are frequent, and during the final stages the caseating process gains the upper hand and breaks through the limiting and protective fibrous tissue spreading the destructive process. The walls of the alveoli are thickened and finally obliterated or filled in, the interlobar connective tissue, especially around the large vessels and bronchi, proliferates enormously and, replacing the parenchymatous tissue of the lung, produces a state of induration through which the dilated bronchi pass.

In all cases of fibroid phthisis the pleura is thickened over the affected area, sometimes attaining a thickness of one-half to three-fourths of an inch. The pleural cavity is adherent and, in the pleural form, obliterated by tough fibrous tissue binding the two surfaces together, and from it other bands of connective tissue are sent forth into the lung which contract and drag along toward the affected side the mediastinum, the diaphragm, and with it the liver, etc.

We are not clear why the tubercle bacilli produce caseation and liquefaction of tissue in most cases, while in others a proliferation of connective tissue is the dominant feature after infection. We know that in many cases of fibroid phthisis we have an additional etiological factor, the inhalation of mineral, animal, and vegetable dust. But on the other hand, the form which will be described as the pleural form of fibroid phthisis is not usually associated with the inhalation of irritating dust, but the causative factor seems to be bacterial, plus the predisposing factors which are operative in the other forms of chronic phthisis.

We are in the dark about these problems. It has not been proven that in fibroid phthisis the tubercle bacilli are of some attenuated strain, or of the bovine type. In many cases of fibroid phthisis in which tubercle bacilli are not detected, Much's granules have been found, thus pointing to bacilli which have lost their acid-fast properties being the cause; but this also requires further study.

Forms of Fibroid Phthisis.—The symptomatology of fibroid phthisis depends on the form of the disease. My experience is in agreement with that of Sokolowski, excepting that I meet with a pleural form in addition to his two forms—simple fibroid phthisis, and fibroid phthisis

with emphysema. The most common clinical form encountered by me is the emphysematous.

The Emphysematous Form.—These patients give a history of cough dating back many years; some state they had coughed as far back as they can recollect. Slight exertion produces dyspnea, and in some slight cyanosis of the lips and finger tips may be observed. They state that they had consulted physicians repeatedly and were informed that the trouble was not of serious import; that it was chronic bronchitis, pulmonary emphysema, etc. Inasmuch as they are able to pursue their occupations, they more or less disregarded the cough, expectoration, dyspnea, etc. During the winter and autumn these patients are usually subject to "colds," "grippe," etc., when the cough is aggravated and persists for several weeks with greater severity than usual.

In some patients, especially those engaged in trades involving the inhalation of animal or vegetable dust, the signs of pulmonary emphysema, as well as attacks simulating bronchial asthma, are apt to come on suddenly in one who had never before suffered from any respiratory trouble. In fact, experience has taught me to look with grave suspicion on emphysema or asthma coming on suddenly in a person over thirty years of age.

During the early stages of the disease, and this may last for many years, the patient, though coughing and suffering from mild dyspnea, pursues his vocation without interruption. Fever is lacking, excepting during an acute exacerbation, or some intercurrent affection. The expectoration is scanty; in fact, the cough is usually dry, or some glairy mucus is brought up after a fit of coughing. A search for tubercle bacilli is usually fruitless. But the dyspnea is annoying and increases on slight exertion.

The general appearance of the patient is that of a healthy person, the *panniculus adiposis* is well preserved, and in those who do not work at hard manual labor, and in women, we may meet with marked obesity. The "fat phthisis," of which we speak elsewhere, is seen almost exclusively in fibroid patients. On the other hand, there are some patients who are more or less emaciated, but they are usually individuals who have never been fat; but even they gain rapidly after the physician urges them to rest and feed up. I have met with some who gained twenty or even more pounds in a couple of months and retained it for years.

The vast majority of fibroid patients have clubbed fingers and curved nails. The most exquisite forms of drumstick fingers may be found among them, while they are not so common among those who suffer from common chronic phthisis.

Many get along fairly well for years without suspecting the real nature of their trouble, until they are suddenly seized by attacks of hemoptysis which may be slight, or quite profuse, but which usually frighten them out of their wits. In some, the hemoptysis recurs at frequent and irregular intervals, and may, at times, be copious, while in

the majority it occurs only rarely and consists only in one or two mouthfuls of blood or streaky sputum. It may appear suddenly while the patient has considered himself in excellent condition. Hemorrhagic phthisis usually is fibroid phthisis, and most patients bear the bleeding very well indeed. I have had patients who were so used to hemoptysis that it no longer frightened them. We meet with many who never expectorated blood.

Well-to-do patients without profuse hemoptysis get along for years without troubling themselves about the cause of their mild cough and dyspnea, unless they apply for life insurance, and after they are rejected for "lung trouble" they promptly consult a physician. It is noteworthy that during the early stages of the disease most of these individuals easily pass the medical examination for life insurance.

Physical Signs.—A physical exploration of the chest usually reveals an emphysematous, or barrel-shaped, chest in those who suffered for years, while in those who have only recently acquired the disease, the thorax may be of normal shape. Careful inspection shows some flattening of the supraclavicular, infraclavicular, and supraspinous fossæ, more marked on one side of the chest, wasted muscles of the neck and shoulder, and shoulder droop on the same side, coupled with lagging and restricted motion. On percussion, defective resonance, or even dulness, is elicited on one side above the second or third rib anteriorly and posteriorly, while below, and all over the opposite side of the chest, the note is hyperresonant, or slightly tympanitic, and the inferior margin of the lung is one or two inches lower than normal and hardly mobile. Narrowing of Krönig's resonant area can easily be made out; in fact, it appears somewhat accentuated because the opposite unaffected apex is larger, owing to emphysema. Auscultation shows feeble breathing all over the chest, while over the site of the dulness the expiratory murmur is harsh and prolonged, at times showing a bronchial timbre. Dry crackles, or rales after cough, may be audible, in others sibilant or sonorous rales are heard all over one side of the chest. During one of the asthmatic attacks, which in some patients are quite frequent, so that they are treated for asthma, we hear wheezing, sibilant and sonorous rales all over the chest, exquisitely simulating bronchial asthma.

Course of the Disease.—These patients get along quite well till they pass middle age. Most of them, if they are under medical care at all, are considered individuals who are troubled with chronic bronchitis, pulmonary emphysema, asthma, etc. But sometimes between the age of forty and sixty, though exceptionally I have seen it in younger individuals, the clinical picture changes. They begin to lose weight gradually and persistently, so that sooner or later they present the unmistakable appearance of the average consumptive in the advanced stages of the disease. The cough becomes more severe and productive of globular and nummular sputum containing tubercle bacilli, elastic tissue, etc. The cyanosis and the dyspnea become

more and more marked, and finally orthopnea sets in with signs and symptoms of dilatation of the right heart which is almost constant at this stage, followed by edema of the lower extremities, hydrothorax, etc. Intestinal and laryngeal tuberculosis are quite common, and contribute to the misery of the patients who finally expire from asystole, or inanition.

The signs in the chest do not differ markedly from those met with in the usual case of far advanced phthisis—signs of cavitation at the apices, as well as of diffuse bronchitis are common. Roentgenography, which in previous stages showed only signs of emphysema with some retraction of one or both apices, now reveals more or less extensive cavities and peribronchial infiltration. Displacements of the mediastinum are more frequent than in common chronic phthisis.

Diagnosis.—In the later stages of the disease the diagnosis is clear and it differs from that of chronic phthisis mainly because of the dyspnea, cyanosis, edema and clubbed fingers, which are not common in the latter condition. In the earlier stages, however, fibroid phthisis is difficult to differentiate from pulmonary emphysema, chronic bronchitis and, at times, from bronchial asthma. The persistently negative sputum is especially perplexing. Errors may, however, be reduced to a minimum by carefully examining the apices in each case of chronic bronchitis and pulmonary emphysema. Whenever the physical signs point to infiltration of an apex, fibroid phthisis is to be thought of. The symptoms and signs of asthma coming on suddenly in one who works in surroundings laden with animal, vegetable, or mineral dust, usually point to fibroid phthisis.

Simple Fibrosis.—These are cases of fibroid phthisis in which the onset, course, and termination of the disease are practically the same as in the form just described, excepting that the symptoms of pulmonary emphysema are lacking. The onset is slow and insidious. The patient is troubled with an occasional morning cough, expectorates little or nothing, and the sputum contains no tubercle bacilli or elastic tissue. There is, however, slight dyspnea on exertion which is often overlooked.

The general condition of the patient leaves little or nothing to be desired. He has no fever, no nightsweats, no anorexia, emaciation, etc. All he complains of, if at all, is that he is subject to "colds," especially during the winter months; of breathlessness, and of hemoptysis, which may be quite a feature in this form of phthisis when occurring often, or is copious. But before, during, and immediately after the hemoptysis there is usually no fever, and convalescence is rapid. In fact, many of the patients feel much relieved after the effects of a brisk pulmonary hemorrhage have passed away. These are the cases which some English authors have described as "arthritic" or "gouty" hemoptysis, because some of these patients, though not all, present some of the stigmata of the arthritic diathesis.

Many of these patients present themselves to their physician, who

makes a careless examination of the chest and, finding no sign of tuberculous infiltration, assured them that the bleeding came from a ruptured bloodvessel in the throat, etc. Thus reassured, they return to work, feeling quite well. However, in many there are signs of active phthisis in one of the apices: Impaired resonance, contraction of Krönig's resonant area, harsh bronchovesicular, or distinctly bronchial, breath-sounds, more or less numerous rales, all localized, circumscribed and persistent above the second rib anteriorly and posteriorly over the supraspinous fossa in one side of the chest. The physician is often amazed to find the patient in such excellent condition for years despite the signs of a distinct and active pulmonary lesion, and is apt to attribute it to chronic apical catarrh.

In other cases the onset is, however, not so insidious. A fairly healthy person is suddenly seized with a pulmonary hemorrhage which may be slight, moderate or, rarely, copious; or he may develop mild fever, nightsweats, cough and expectorate sputum containing tubercle bacilli. A physical exploration of the chest shows a typical lesion of moderate extent. Inasmuch as for several weeks the patient presents most of the symptoms and signs of progressive phthisis, even hectic fever, nightsweats, emaciation, etc., a grave or doubtful prognosis is rendered.

But slowly the condition of the patient begins to improve; the fever abates, the cough is ameliorated or ceases altogether, the appetite improves and the patient gains in weight considerably, so that in a few months his weight exceeds that found before the onset of the disease. He considers himself cured. But a physical examination of his chest shows distinct and unmistakable signs of a smouldering tuberculous lesion in one apex; in fact, the signs of active disease are there and tubercle bacilli may be found in the sputum. Feeling well, the patient reënters his occupation and works quite efficiently, believing that the physician who declared him still actively tuberculous is an alarmist. I have had patients of this class who have been doing well for years, and who came around to the office to "prove" it to me. Many are of the class who were admitted as advanced cases, and then discharged from sanatoriums as improved, or even "unimproved," and inquiry in later years shows that a large proportion remain in good condition and working, except for more or less pronounced dyspnea which annoys them.

After some years the symptoms are gradually aggravated, they complain they have "caught a new cold," which is difficult to cure. The cough is persistent and exhausting, the dyspnea distressing, and they begin to lose in weight and strength progressively, presenting clearly the characteristic clinical picture of chronic phthisis with its usual complications, plus dilatation of the right heart, dyspnea and orthopnea. Physical exploration of the chest shows the usual clinical picture of cavitary phthisis, but there is in addition bronchitis, which is unusual in chronic phthisis. It differs, however, from chronic

phthisis by the fact that fever is lacking, or, at most, some insignificant elevation of temperature is noted at times. No nightsweats are present, or only slight, at the end of the disease.

Pleural Form of Fibroid Phthisis.—In the pleural form of fibroid phthisis, the patient usually gives a history of an attack of pleurisy with effusion, from which he has recovered after a longer or shorter illness, the fluid having been absorbed spontaneously, or was aspirated. But ever since he has remained with a dry, hacking cough, productive of little or no sputum, and in spite of the great care he has been taking of himself, he has not succeeded in recuperating completely. Dyspnea is marked and increasing steadily in intensity. In many cases the cyanosis of the fingers and face is very pronounced.

During recent years I have met with some cases of this type following artificial pneumothorax. A pleural effusion was slow in disappearing, and the gas inflations had to be discontinued. But the patient kept well on the road to recovery, remaining with a pleuropulmonary tuberculous lesion.

Examination shows distinct immobility of the lower half of the side of the chest in which the effusion had taken place; some retraction of the chest wall and scoliosis, or kyphoscoliosis. Mensuration shows that the affected side has fallen in—the circumference being smaller than the unaffected side by more than one inch. Vocal fremitus is absent over the area. On percussion we find dulness, at times even flatness not unlike that over pleural effusion, which is at once suspected. This is apparently confirmed by the absence of the vocal fremitus and of any breath sounds, while in some we hear distant tubular, or even cavernous breathing. There may be no adventitious sounds, but occasionally some medium-sized or large, moist and consonating rales and gurgles are audible during both phases of respiration. At times, distinct friction sounds, grating, and grunts are heard. Over the upper half of the chest signs of a tuberculous lesion are usually found—bronchial breath sounds, moist rales, and impaired resonance. In many signs of a cavity are elicited.

On the unaffected side signs of pulmonary emphysema are found—hyperresonance and the inferior margin of the lung extends two to four inches lower than on the opposite side, owing to emphysema, and the pulmonary retraction and upward displacement of the diaphragm on the affected side accentuate it. Anteriorly, the border of the unaffected lung extends well over the sternum.

The heart is almost invariably dislocated toward the affected side, which serves as a good sign of differentiation from pleural effusion with which it may be confused, because in effusions the dislocation is invariably toward the unaffected side, if at all. In left-sided lesions we may find the apex as far out as the axillary line and one or two interspaces higher than the normal; in right-sided lesions the apex may be found at the xyphoid cartilage, or even farther to the right. It is in these forms of phthisis that acquired dextrocardia is at times

found. It is due to traction of the heart by fibrous bands in the right pleura and lung and also to the pressure exerted by the vicariously emphysematous left lung. The shrinkage, as well as the fibrous bands in the lungs, also drag the diaphragm upward and when the right side is affected, the liver is also elevated. In the left side the stomach may be elevated along with the diaphragm. Pulmonary retraction in the left side also exposes the heart and brings it near the chest walls, where we may see it pulsating. These conditions may be made out by careful percussion, but in many cases the aid of roentgenography is necessary to clear up mooted points.

There are other clinical peculiarities which should be mentioned. Fever is usually absent throughout the course, excepting when due to some intercurrent affection. When we find a persistent elevation of temperature we may look for some complication, especially an infiltration of the opposite, hitherto unaffected lung. The cough, which was moderate for a long time, in some cases for years, becomes more and more severe and the amount of sputum brought up may be enormous. Both, the cough and the expectoration, may be influenced by posture—the patient coughs more when lying on one side, and is somewhat relieved when turning on the other side, just as in bronchiectasis. This, however, gives no clue as to which side is affected. The sputum contains tubercle bacilli in large numbers and is at times fetid, which is rare in other forms of phthisis.

Hemoptysis, which is very frequent in other forms of fibroid phthisis, is less often encountered in the pleural form. But when occurring, it is apt to last for days or weeks, and at times it is copious. I have seen cases in which it was the cause of death of patients who were otherwise getting along very well. Most of these patients have clubbed fingers and curved nails.

Dyspnea, which is a feature of all forms of fibroid phthisis, is more severe in this type because of the loss of lung tissue and the displacement of the heart. In fact, I have seen many cases in which the lesion in the lung was practically healed, or at least distinctly inactive, yet the dyspnea was severe and unbearable. Another feature is cardiac palpitation, especially in left-sided lesions, which is apt to be so severe as to make life unbearable.

In the terminal stages signs of cardiac dilatation set in—edema of the lower extremities, enlargement of the liver, cyanosis, etc., and the patient dies from asystole. In many cases complications are responsible for the final outcome—hemorrhage, which was already mentioned, inanition due to laryngeal tuberculosis with dysphagia, amyloid degeneration of the various visceral organs, etc. Tuberculosis of the previously unaffected lung may bring about a rapidly fatal course of the disease.

I have observed that some of these cases, tuberculous in origin as they are, become purely bronchiectatic. The tubercle bacilli disappear from the sputum, but the patient continues to cough and expecto-

rate large quantities of sputum which shows all the characteristics of sputum in bronchiectasis; in fact, the course is that of non-specific bronchiectasis after this occurrence.

Prognosis in Fibroid Phthisis.—As regards duration of life, fibroid phthisis, though an active tuberculous disease and hardly ever cured, is more favorable than the other forms of phthisis, excepting abortive tuberculosis. It is among the fibroid patients that we find individuals who have been tuberculous for years. I have some who have lasted for twenty-five years, and Sokolowski reports one who lasted for more than forty years. While they are always ailing, many are still fit to pursue their vocations, and I have among my clientele some who have worked quite hard without long interruptions.

In fibroid phthisis, the reparative processes of Nature are more active than the destructive tuberculous, and the patients are shielded from the extension of the caseating and softening processes, the fibrous tissue usually forming a wall around the lesion limiting its progress and preventing the absorption of toxins, as is evident from the absence of fever, etc. Because of the pleural adhesions, the patients are shielded from such complications as spontaneous pneumothorax, which never occurs among them. When in my hospital practice I find a fibroid patient presenting the symptoms of spontaneous pneumothorax, it is soon clear that the rupture occurred in the lung which had been unaffected but recently showed a new lesion.

CHAPTER XXIII.

ACUTE FORMS OF TUBERCULOSIS.

As in other infectious diseases, there are observed in tuberculosis acute, malignant, or fulminating forms which run a shorter, and almost invariably fatal course. They are relatively rare, as malignant scarlet, measles, typhoid, etc., are rare. Every practitioner meets with these acute cases, and the laity is well aware of their existence. When tuberculosis makes its appearance in a member of a family anxious inquiries are made to ascertain whether it is not "hasty," or "galloping consumption," the names under which acute tuberculosis is commonly known. Pathologically, the lesion is practically the same as that of the chronic forms of the disease, considering that there are no two cases of phthisis in which the anatomical changes are exactly alike, but clinically it manifests itself by a more rapid course, the patient lasting as many months with the acute form as years with the chronic forms. *Acute pulmonary tuberculosis may be said to be active chronic phthisis without the remissions and ameliorations characteristic of the course of the latter affection.*

It is unnecessary to enter into hair-splitting distinctions of the pathological and clinical types of acute pulmonary tuberculosis described by some authors. In practice we meet mainly with two types of the disease: The lobar pneumonic type—acute pneumonic phthisis, and the lobular, or bronchopneumonic type. In the former the patients are usually adults, while the latter attacks mainly infants and very young children, and adults only at the terminal stages of chronic phthisis.

Between the two extremes—chronic and acute phthisis—there are many gradations; some are very acute, the patient being carried off within one or two weeks; some are subacute, lasting for two to four months, others even a year, but without any remissions in the progress. Then there are acute exacerbations during the course of chronic phthisis which are anatomically and clinically of the same character as the acute or subacute forms and often bring hitherto hopeful cases to a speedy termination. I have also met with cases which began acutely and kept up in that manner for several weeks, but suddenly, or by degrees, took a turn for the better, and the patient passed through the course of chronic phthisis subsequently.

Etiology.—The factors operative in causing an acute and malignant evolution of phthisis in some cases, while in the vast majority it is chronic, slow, and more or less benign, are not clear. From a careful

study of the cases met in practice it appears that the general condition of the patient before the onset of the disease has no influence in this direction. In fact, it appears, as will be shown later on (see p. 584), phthisis in those who suffered from scrofula, osseous, or glandular tuberculosis during childhood, or who are descended from tuberculous stock, is more likely to run a slow, sluggish course. On the other hand, we very often meet with acute phthisis in persons who have no hereditary taint, athletic individuals, who have been in excellent condition, and only rarely in the weakly and decrepit, excepting tuberculous bronchopneumonia in infants.

The problem whether these acute cases are invariably due to more virulent strains of tubercle bacilli has not been solved, though there appears to be no evidence in favor of such a view. Some authors have held that acute phthisis is caused when a tuberculous cavity, or a caseating gland, breaks through into the lung, disseminating the secretions containing bacilli, but this is negatived by the fact that we meet numerous patients who never coughed before the onset of the acute disease.

It appears that individuals who have never before been in tubercle-laden surroundings are more likely to develop acute phthisis when infected primarily after they have passed the age of childhood, as we have already shown (see p. 73). The same "virgin soil" is presented by infants: when they are infected with tuberculosis they very often suffer from the acute forms of the disease, and so do adults hailing from rural districts where they have not met with tuberculosis, so that if infection takes place it is primary. The explanation of these phenomena has been discussed in a previous chapter.

ACUTE FORMS OF PULMONARY TUBERCULOSIS.

Acute Pneumonic Phthisis (Galloping Consumption.)—The anatomical changes are those of pulmonary tuberculosis but the process of caseation and liquefaction gains the upper hand, not being limited by the conservative process of fibrosis which is a strong feature in chronic phthisis; little or no connective tissue is formed to localize the lesion. Usually the greater part of a lobe, or a whole lobe, is affected. The parenchyma is transformed into a solid, caseous, or gelatinous mass within which there can often be found a focus representing an old lesion. The destruction of lung tissue goes on at a rapid pace, and within a short time more or less extensive excavations may be formed. But these excavations are not surrounded by a connective-tissue wall; all around them is caseated lung tissue. In many cases, however, death supervenes before softening has had time to set in to sequestrate the affected part of the lung. We may find scattered tubercles or caseous nodules all over the affected lung and also in the other, as well as on the visceral pleura, but pleural adhesions are rare.

Symptomatology.—The disease is mostly seen in adults between twenty and forty years of age. The onset and symptoms during the first few days are akin to those of lobar pneumonia. In fact, most of the cases of chronic phthisis which are said to have begun as lobar pneumonia are acute pneumonic phthisis which was not recognized as such at the onset of the acute stage.

As given by the patients, the onset is nearly always acute. After some alleged exposure there was a chill, fever, pain in the chest, cough, etc. But a careful inquiry elicits that while the acute symptoms have come on suddenly, the patient has for weeks, perhaps for months, felt out of sorts; was unable to perform his usual work without fatigue; in fact, he has coughed, expectorated and may have had some night-sweats. But these symptoms were not sufficiently pronounced to cause alarm; even if he has consulted his physician he may have been told that his troubles were trifling. *This long prodromal stage is of great diagnostic importance, and will often aid while attempting to differentiate acute pneumonic phthisis from lobar pneumonia.*

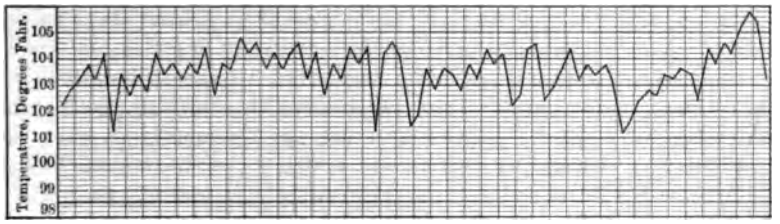


FIG. 87.—Temperature curve in acute pneumonic phthisis.

With the acute symptoms the patient is laid up in bed. The dyspnea is marked from the beginning, and may be paroxysmal. The pain in the side is mild, and only rarely as acute as in pneumonia or pleurisy, or may be altogether lacking. Cough is nearly always annoying; it may be severe, incessant, and exhausting. At first dry, it slowly becomes productive and the sputum is at times rusty and viscid, adhering to the sides of the vessel like in lobar pneumonia. But in most cases it is mucopurulent, frothy and easily brought up. In some cases it is sanguineous, at times repeated, small, true hemoptyses take place, and the disease may begin with a profuse pulmonary hemorrhage. When softening and excavation take place, which occur quite soon, the sputum is of the same character as that of chronic phthisis, excepting that it is more often green in color. In the beginning repeated microscopic examinations do not reveal any tubercle bacilli, and, because pneumococci are quite frequent, the diagnosis is very difficult. Only after the disease has lasted for a couple of weeks, and very often much later, when we may be thinking that we are dealing with an unresolved pneumonia, tubercle bacilli are discovered in the sputum.

Weakness, anorexia, emaciation, and fever are very strong clinical features in the evolution of the disease. The weakness may be so severe that very early in the course of the disease the patient is unable to sit up in bed, or to breathe deeply for the purpose of auscultation. When examined he falls back in bed exhausted, pale and cyanosed. This asthenia is not seen in the average case of lobar pneumonia. With the anorexia, which may be pronounced from the very beginning, emaciation goes hand in hand. Even in the cases in which the appetite is somewhat retained, the emaciation is very early and pronounced, and out of proportion to the fever and anorexia. It usually proceeds rapidly and often frightfully, so that within a few weeks a normally built man is reduced to a skeleton. Wasting is particularly quick in the muscles of the chest. More or less copious pulmonary hemorrhages may occur at any time. Some begin with a hemorrhage, as has already been stated. In others it occurs during the course of the disease, and now and then we meet with a case of acute pneumonic phthisis which succumbs to a profuse hemorrhage.

In the beginning the fever is of a continuous type, like in lobar pneumonia, though some authors have described pneumonic phthisis without high fever, which I have never met in my practice. But this is rare during the first few weeks when the temperature curve exquisitely simulates that of lobar pneumonia, but during the second week, when we expect defervescence, we are disappointed. Instead of this, the fever becomes intermittent, or hectic, with morning remissions to normal or even below, and afternoon rises to 103° or 104° F., and accompanied by copious nightsweats. The pulse is rapid, small and feeble, and the blood-pressure low. The full, vigorous pulse of lobar pneumonia is never found.

Physical Signs.—Physical exploration of the chest often shows the signs of typical lobar pneumonia. There is impaired resonance or dulness over the upper part of one side of the chest above the third rib. But instead of the harsh tubular breathing, which is characteristic of pneumonia, we usually perceive diminished and, in some cases, complete absence of breath sounds, which are replaced by moist, subcrepitant rales. The crepitation of pneumonia is only rarely audible. With the advance of the lesion the dulness becomes more pronounced and the respiratory murmur may be altogether abolished, or bronchial breathing may become audible coupled with small, and medium-sized moist rales. In acutely progressive cases signs of excavation may be found within four weeks; but this is rare. In many cases the lesion is centrally located, and the physical signs are indefinite. The writer has seen a case in which competent clinicians could not localize the lesion for about three months, when finally physical signs appeared over the upper lobe of the right lung.

Course.—In most cases the acute symptoms persist for two or three months, the lesion softens, extensive excavations may form and the patient finally succumbs to asthenia. In some the process is of shorter

duration; I have seen cases in which death occurred in less than three weeks. In rare instances the disease is acute for four to six weeks, when an improvement in the general condition takes place and, with more or less extensive excavation in a lung, the patient becomes a chronic consumptive and the disease may even be arrested in time, which is, however, very rare. In some the toxemia is very severe, and the patient succumbs within two or three weeks, even before softening has taken place. The prognosis under the circumstances is very grave, the average duration of the fatal cases, and they are in the vast majority, is about six weeks, dying from toxemia and exhaustion. I have seen several cases in which the end came through a brisk pulmonary hemorrhage.

Differential Diagnosis.—It is often very difficult to differentiate acute pneumonic phthisis from lobar pneumonia, especially during the first two weeks of the ailment. Mistakes may be avoided by carefully inquiring for premonitory symptoms of tuberculosis preceding the acute onset, such as anorexia, emaciation, weakness, mild cough, nightsweats, etc., which are frequent in acute phthisis, while in lobar pneumonia the patient is stricken suddenly when he feels in the best of health. In fact, in *apical pneumonia, and in pneumonia following an atypical course, acute tuberculosis is always to be thought of.* The absence of pain in the side, the late arrival of true bronchial breathing, the hemoptysis, etc., may all lead to a diagnosis, or at least a suspicion of acute phthisis. An irregular temperature curve, mild dyspnea, severe pallor, low leukocyte count, absence of pneumococci from the sputum, and a strong diazo-reaction may also be considered. Of great importance in favor of acute phthisis is yellow or green sputum. Tubercle bacilli are conclusive evidence, but they are only rarely found before the end of a month. On the other hand, in lobar pneumonia complicating pulmonary tuberculosis, we may find tubercle bacilli in the sputum. Several cases of this type have come under the writer's observation, and a diagnosis of acute pneumonic phthisis was made, and within a week or ten days the pneumonia ended by crisis, while the tuberculous disease subsequently pursued its course unaffected by the pneumonic complication. However, these complicating pneumonias, as a rule, attack one of the lower lobes, and this fact should be considered in all cases. During the first week the emaciation is negligible in pneumonia, irrespective of the acuteness of the symptoms, while in phthisis it is immediately pronounced; nightsweats, weakness and edema of the lower limbs are frequent. The crisis, which is sure to come before the fourteenth day in the vast majority of cases of pneumonia, will clear up doubtful cases.

Especially difficult is the diagnosis of pneumonic phthisis in aged persons, in whom it may occur without much fever and other general symptoms, and only positive sputum can decide.

Tuberculous Bronchopneumonia.—Etiology.—The anatomical changes in tuberculous bronchopneumonia are those of pulmonary tuberculosis,

excepting that the lesion is not localized in one apex, or one lobe, but disseminated all over one or both lungs in which there are distributed caseous nodules which vary in size from that of a pin-point to that of a walnut. Some authors have been inclined to attribute the wide dissemination of the lesion, as well as the acute course of this form of tuberculosis, to mixed infection with tubercle bacilli and pyogenic microörganisms. This, they believe, is confirmed by the fact that it very often follows other infections such as measles, whooping-cough, influenza, typhoid, etc., showing that the patient had harbored a tuberculous process before, but with the addition of a new infective agent his vitality was reduced and the tuberculous process allowed to spread all over the lungs. But against this view may be brought forward the numerous cases in which mixed infection can be positively excluded. Moreover, as will be shown elsewhere, in cases in which typhoid fever or influenza is supposed to have preceded the tuberculous disease, careful investigation usually reveals that the acute initial symptoms were those of tuberculosis, but an erroneous diagnosis was made of some other infection.

In most cases it appears to be the result of the wide dissemination of the contents of a tuberculous cavity in the lungs, or the perforation of a tuberculous lymph node, the contents of which are aspirated, carried all over the bronchial tree and take root in various parts of the lungs. In infants, among whom this form of the disease is very common, it may be due to a primary massive infection with tubercle bacilli; the body possessing no immunity through previous infection, the result is the same as when a guinea-pig is infected. In adults, we also meet it in women after childbirth, in tuberculosis with diabetes and alcoholism, etc., and more commonly, as a terminal phenomenon of chronic phthisis when the resisting powers are at low ebb, and immunity acquired by the existing lesion is lacking.

Symptoms.—Tuberculous bronchopneumonia in adults is usually found in patients who have been tuberculous for some time. In those in whom it appears to be of sudden onset, careful inquiry elicits the information that the patients have been ailing for some time with symptoms highly suggestive of tuberculosis. In fact, it is often a complication of chronic phthisis: A patient who has been doing fairly well suddenly develops acute symptoms without any special cause; more often after a surgical operation in which a general anesthetic was employed. Tuberculous women are, at times, the victims soon after childbirth.

The clinical picture is that of an acute infectious disease with pronounced toxemia. The onset is sudden, often with a chill, fever, backache, cough, expectoration, etc. The fever is usually high—103° to 104° F. is not uncommon—and in children it may be even higher. The temperature curve is not characteristic; in fact, it may be stated that its peculiarity is its irregularity. In many cases it is continuous with slight remissions, but in others it is intermittent,

with chills before each rise. During the terminal stages it is usually hectic. The sweats are profuse and exhausting, the pulse feeble, small and rapid, 120 to 150 is not rare; the dyspnea is marked—40 to 60 per minute are very often counted, and cyanosis is a frequent feature. Graves spoke of "acute tubercular asphyxia."

The intensity of the cough is variable: In some patients it is severe, painful, paroxysmal, and may provoke vomiting. While occasionally the cough is mild, in most cases it is more severe than in chronic phthisis. At times expectoration is absent or scanty, but usually it is more or less abundant, often purulent, and, with the advance of the disease, nummular; yellowish-green balls are brought up. Tubercle bacilli are found in most cases.

Hemoptysis is frequent in adults and may be quite copious; many cases begin with pulmonary hemorrhage.

The appetite is rarely fairly well retained, but in most cases this, as well as the digestive functions, is impaired; many have to be coaxed to take some nourishment. Emaciation proceeds at a rapid pace.

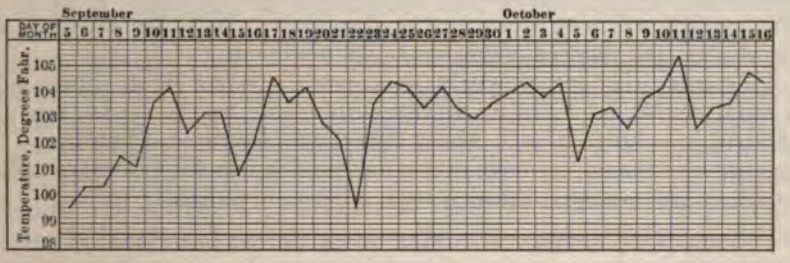


FIG. 88.—Temperature curve in tuberculous bronchopneumonia.

Because of the flushed face it is at times not appreciated at first sight, but when the bedclothes are removed, the marked wasting of the subcutaneous tissues and muscles of the chest and extremities presents a frightful picture, especially when it is considered that it may have been consummated within a few weeks.

Physical Signs.—The physical signs vary according to the nature of the anatomical changes in the lungs. In the beginning they may be obscure and misleading. In most cases the note elicited on percussion is hyperresonant all over the two sides of the thorax; localized dulness is found only later when some of the disseminated tubercles have become confluent. Auscultation shows either feeble breathing or harsh bronchovesicular breath sounds all over the chest, coupled with sibilant and sonorous rales. With the advance of the disease, which may be within but one or two weeks, we find localized areas, not necessarily in the apex, especially in children, of consolidation with bronchial breathing and moist subcrepitant rales which soon change their character when excavation takes place and the usual signs of a cavity can be made out. In many cases, notably in children,

signs of diffuse bronchitis are found all over the chest, while in others the toxemia is so severe that the patient succumbs before definite changes in the resonance and breath sounds have developed.

Complications.—Among these may be mentioned pulmonary hemorrhage, which may be fatal; intestinal tuberculosis, tuberculous meningitis, and general miliary tuberculosis.

Diagnosis.—The diagnosis is very difficult in the initial stages, particularly in children, among whom it must be differentiated from postgrippal bronchopneumonia, and sputum is not available for microscopical examination. In adults it is usually more easily diagnosticated. We find in patients who have been tuberculous for some time that after a hemorrhage, surgical anesthesia, pregnancy, etc., the symptoms suddenly take a sharp turn and galloping consumption follows. It is always to be borne in mind that *when in a person who never before had emphysema, and who has no barrel-shaped chest, symptoms and signs of emphysema suddenly make their appearance accompanied by acute constitutional symptoms such as fever, cough, dyspnea, cyanosis, nightsweats, etc., acute phthisis is to be thought of.* The sputum will soon clear up the diagnosis. With the advance of the disease the physical signs are easily made out.

Prognosis.—The prognosis is very grave. Some acute cases run a rapid course, terminating fatally within four to six weeks, and in children in a shorter time. Many cases linger for three or four months, and die of asthenia. I have met some cases in which the disease came to a halt and assumed the character of chronic phthisis.

ACUTE MILIARY TUBERCULOSIS.

Pathogenesis.—When from an existing tuberculous focus anywhere in the body a large number of tubercle bacilli are released into the blood- or lymph-stream, a bacteremia results, the microörganisms settling in the various visceral organs producing small tuberculous nodules. In this form of acute tuberculosis most of the viscera are affected, while in the acute forms of pulmonary tuberculosis, which have just been described, or in the various forms of tuberculosis of the glands, serous membranes, bones, or joints, only the affected tissues bear the brunt of the infection.

Acute miliary tuberculosis is hardly ever primary; in nearly all cases there had existed a localized tuberculous focus which may have been small, hardly detectable at the autopsy, and which may have given no symptoms during the life of the patient. But when a bloodvessel passing through, or in contact with, a caseated lymphatic gland, or a caseated area of any tissue, erodes; or when a tubercle involving the wall of a vessel softens, and produces ulceration of the intima, large numbers of tubercle bacilli are set free into the circulating blood. Similar effects will be produced indirectly when the thoracic duct is

affected by tuberculosis, and bacilli enter through the subclavian vein into the general circulation.

It is, however, rare that a caseated tuberculous area should break into the circulation directly. The most common mode appears to be the formation of tubercles in the walls of a vessel, especially the pulmonary vein, or the thoracic duct. Benda[1] found metastatic tuberculous endangeitis more common than periangeitis. When these tubercles soften, breaking through the endothelial lining, a constant stream of tubercle bacilli enters the circulation. Its occurrence is comparatively infrequent, because tuberculous endangeitis is relatively rare owing to the fact that in the usual tuberculous lesion the blood-vessels within, and in the neighborhood of, old foci are thickened as a result of the inflammatory reaction, their lumen is obliterated, and thus erosion and passage of tubercle bacilli into the blood stream is prevented. Otherwise the vast majority of cases of tuberculous infection would implicate bloodvessels and lymphatics in and adjoining the lesions, and acute miliary tuberculosis would result.

It must, however, be emphasized that a tuberculous bacteremia is not sufficient to produce acute miliary tuberculosis; excepting in cases of primary massive infections which occur in infants, as has been shown elsewhere. In adults with chronic tuberculous lesions, among whom primary tuberculous infection is, as a rule, out of the question for reasons already stated, tubercle bacilli are very often found in the circulating blood, yet no miliary tubercles are formed in the visceral organs. It seems that before the body can be overwhelmed by tubercle bacilli and permit them to produce miliary tubercles in the various viscera, some factors which reduce the natural resisting forces must be at work, the blood must have lost its normal bactericidal powers, to permit the bacilli in a virulent state to settle anywhere they please. In the usual case of local tuberculosis the bactericidal action of the blood is effective in either killing the organisms, or at least in attenuating their virulence, so that even animal inoculation of blood containing acid-fast bacilli proves harmless (see p. 285).

What these etiological factors are, we do not know, and when they are elucidated, the most important problems in the pathogenesis of tuberculosis will be cleared up. In many tuberculous patients with active disease, acute miliary tuberculosis appears as a terminal phenomenon. In others, with healed or arrested lesions, symptoms of acute miliary tuberculosis appear leading to a fatal issue after chilling the body, after surgical operations, especially for some extra-thoracic tuberculous lesion, or without any obvious exciting cause. In hospitals for tuberculous patients there are at times to be observed "epidemics" of acute miliary tuberculosis. This is especially seen during changeable meteorological conditions, or when intercurrent infections are common. It may also occur after a pleural effusion is rapidly absorbed, or aspirated.

[1] Ergebn. d. allg. Pathol., 1900, **5**, 447.

Evidently because tuberculous infection is primary in infants, when massive, it may result in acute miliary tuberculosis; it usually follows some intercurrent infection, as measles or pertussis. But it is not as rare in adults as some believe. A large proportion of fatal cases of chronic phthisis are carried off by symptoms and acute miliary tuberculosis which make their appearance at the end. It has been suggested that the accessible and wide lymphatic system of infants offers better opportunities for the bacilli to enter the lymph glands, and after these soften, they pass into the thoracic duct, finally reaching the general circulation and then disseminating throughout the body.

Pathology.—Miliary tubercles are found in most of the visceral organs. The lungs, as the *locus minoris resistentiæ*, contain more than other organs, but the spleen, liver, kidney, also the thyroid, bone-marrow, the heart, meninges, peritoneum, choroids, etc., may be studded with miliary and submiliary tubercles. These nodules are of various sizes, according to their age and density of conglomeration; young ones are small, hardly visible, at first gray and transparent, but later many adjoin one another and caseation begins, when they appear yellowish. In each organ tubercles of various ages may be discerned owing to repeated migration of bacilli from the original focus which settle wherever they find a suitable soil. The surface of the affected organs, as well as the large serous membranes, may be found sprinkled with these nodules. In most cases the old original tuberculous lesions, the source of the bacilli which overwhelmed the body, may be discovered in the lungs or glands. In adults, in whom acute miliary tuberculosis was a terminal phenomenon of chronic phthisis any kind of tuberculous changes may be found in the lungs, pleura, peritoneum, larynx, intestine, etc.

Symptoms.—The manifold origin, and different localization of the main clusters of tubercle within the visceral organs result in a variegated symptomatology of acute miliary tuberculosis. In cases which the tubercles are scattered equally among these organs, the symptoms are not unlike those of other forms of acute general infections, as septicemia, or typhoid fever; in fact a typhoid form of acute miliary tuberculosis has been described. In cases in which tubercles are sprinkled over the meninges, cerebral symptoms predominate and may overwhelm the clinical phenomena. We thus have a meningeal form of miliary tuberculosis. In some cases the lungs bear the brunt of the infection, pulmonary symptoms are then in the ascendency, producing the pulmonary form of the disease. However, it must be emphasized at the outset that mixed forms are very frequently met with, or one form passes into another; the typhoid and pulmonary forms very often present symptoms of the meningeal form at the end. Consequently, no sharp line of demarcation can be drawn between these three clinical forms of the disease, and it is merely for descriptive purposes that this division is followed.

The Typhoid Form.—The onset may be sudden without any premonitory symptoms. The patient may have considered himself well, never having known of any tuberculous lesion in his body, or he may have had some slight cough for years which was known to be of tuberculous origin, but he has been pronounced cured. He may have been tuberculous for months or years, and properly treated, the disease pursuing a favorable course, etc., when suddenly, after some exposure, or without any assignable cause, he has a chill, his temperature rises, and he presents symptoms of a profound intoxication. The fever rises to 103° F., or higher, within one or two days, the pulse is rapid, small, and soft, the blood-pressure low, and other symptoms of toxemia make their appearance—dry, coated tongue, anorexia, dyspnea, etc. Delirium may be an early symptom, or it may appear toward the end. While constipation is not rare, in some cases there is annoying diarrhea and this, with scattered roseola over the body, and a large spleen, point to typhoid fever which is often diagnosed especially in patients who had no symptoms of tuberculosis before the onset of the acute process.

Physical exploration of the chest may prove negative, or signs of a disseminated bronchitis may be elicited. But a careful study of the symptoms and course shows the real condition. Thus, a characteristic fever curve of typhoid is lacking; instead of high continuous fever, we may have a remittent curve, which later becomes intermittent. In many cases the fever curve is altogether irregular, for a few days it is of one type, then of another. Rarely the "reverse" type (see p. 222) is observed. The emaciation is rapid and pronounced, dyspnea and cyanosis are more severe than in the average case of uncomplicated typhoid. Then cerebral symptoms make their appearance: rigidity of the neck, differences in the pupils, evidences of palsies, especially of the cranial nerves, etc., and within two or three weeks a fatal termination occurs.

The Pulmonary Form.—Here the conglomeration of tubercles predominates in the lungs; not only one or more lobes are mainly affected, as is the case in acute pneumonic phthisis, but nodules are scattered all over both lungs. The symptoms are those of profound toxemia with signs of pulmonary disease. The patients may have coughed for some time, or may have been known as tuberculous for months or years. In rare instances no such history is obtained. The temperature rises, and within two or three days it reaches 104° F., or higher. In old persons it may be lower, and in some cases it hardly exceeds 99.5° F. With the fever there is profuse sweating, not only during the night, but also during the day. In some instances the disease begins with a chill, and chilly sensations may occur several times a day during the course of the disease, the chills being followed by a rise in the temperature, and then by profuse sweating. In some cases, especially at the end, there is observed an intermittent temperature, with subnormal, or even collapse, temperature in the early morning hours.

Symptoms referable to the respiratory system are present in nearly all cases. In some instances cough may be altogether lacking during the first week of the disease, but in most there is a dry hacking cough, and paroxysmal attacks of great severity are not rare. Usually the cough is unproductive early in the disease, excepting in those in whom the acute process is superimposed on an old phthisical condition. The sputum brought up is not characteristic and, as a rule, does not show the presence of tubercle bacilli. Streaks of blood may be observed in the sputum, and rusty sputum, without any evidences of pneumonia, is seen in rare instances. In patients with old tuberculous lesions there may occur copious, or even fatal, pulmonary hemorrhages, while in those in whom the acute process complicates chronic phthisis acute miliary tuberculosis may follow a brisk hemorrhage. But this is rare.

Dyspnea and cyanosis of the lips and finger tips are seen in most cases very early, and this is important diagnostically. The breathing is superficial and rapid, 50 to 80 per minute; in some cases orthopnea is observed. In patients without pronounced old lesions, exploration

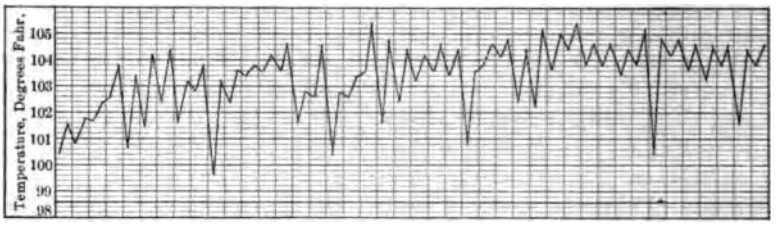


FIG. 89.—Temperature curve in acute miliary tuberculosis.

of the chest may reveal nothing definite, or some small, moist rales may be audible over any part of the lungs; in others sibilation in either, or both, sides is heard. Percussion does not bring out any localized areas of consolidation, but tympany may be found all over, in on some parts, especially the upper third of the chest, dulness with a tympanitic overtone, owing to the relaxation of the parenchyma because of the dissiminated nodules within them. Here and there some moist rale is made out, and friction sounds may be audible over the lower parts of the chest. In many cases symptoms and signs not unlike those of pulmonary emphysema are elicited. On the whole, there is a striking discordance between the severity of the constitutional symptoms and the paucity of the physical signs, and this is of diagnostic significance.

With the advance of the disease, which may continue for four to eight weeks, and rarely longer, symptoms of the typhoid form, and of meningeal irritation, make their appearance, and the end comes either from exhaustion and collapse with a subnormal temperature, or, more commonly, with high fever, delirium and meningeal symptoms.

Meningeal Form.—This is most frequently seen in children, but in adults with known tuberculous lesions in any organ which have lasted for some time, it is a not very uncommon mode of death. About 5 per cent of patients with pulmonary tuberculosis succumb finally to tuberculous meningitis. Metastasis in the meninges is more likely to occur in cases of extra-pulmonary lesions, active or latent. Thus, as is shown elsewhere, it is very frequent in genito-urinary tuberculosis in adults, and in children with osseous, glandular and articular tuberculosis. In infants, however, it often follows tuberculous bronchopneumonia. But here also the intrathoracic glands are severely affected, as a rule. The general infection occurs hematogenously, but it may also take place lymphogenously.

Symptoms in Children.—In children the onset may be abrupt, with persistent vomiting or convulsions. But in such cases, if carefully looked for, a chronic tuberculous process in some glands may be found, though it may not have been inconsistent with apparent health of the child. More frequently it was known for weeks or months that the child was sick. It may have been coughing, but this was attributed to some mild disorder in the throat or bronchi; it may have had some recognized tuberculous gland on the neck, or within the chest, or a tuberculous lesion in a bone or joint. But all this was considered trifling. The child, however, kept on losing ground, its appetite has been poor, and it has lost, or failed to gain, in weight. Its mental state has become rather peculiar, it has been out of sorts, refused to play, sought seclusion, complained of headache, avoided bright light and loud noises. Constipation is the rule, but in some the reverse is true. In few, attacks of vomiting, at times of the projectile type, may be observed at this stage. Careful thermometry will reveal a mild subfebrile temperature.

This prodromal stage may last for several weeks, but in most cases within two or three weeks, symptoms pointing to the cerebral nervous system make their appearance, such as a slight facial palsy, a squint, cutaneous hyperesthesia, erythematous blotches, dermographism, stiffness of the neck, etc. In some instances palsy of a limb is noted at this stage. The child becomes drowsy, responds only when strongly urged. The pupils may be normal, or contracted, but they respond to light. In some, nystagmus is seen at this stage. The reflexes are exaggerated. Spastic contraction of the muscles, especially those of the spine, is common, the neck is rigid and drawn back, and later opisthotonos may occur. Kernig's sign is present in nearly all cases. Later on the spasticity may become more accentuated on one side, and there may occur paralysis of an arm or leg, and of some of the cranial nerves, especially the facial and oculomotor. The pupils now cease to respond to light, and inequality is not uncommon. Delirium, frequent in adults, is rare in children; they are usually in stupor from which it is hard, or impossible, to arouse them. Convulsions may now become more and more frequent, or coma supervenes. The vaso-

motor disturbances now become more and more pronounced, *tâche cérébrale* is hardly ever absent. The pulse, which may have been rather slow in the beginning, now becomes rapid and irregular.

As is the case in adults, remissions may occur, and false hopes are thus engendered when the stuporous child wakes and acts quite normally for a few hours, a day or two. But a relapse is sure to occur, the symptoms return with greater severity, and within one to three weeks the stupor passes into coma from which the child cannot be aroused and, with Cheyne-Stokes breathing, convulsions, hyperpyrexia, etc., the child expires.

Symptoms in Adults.—Many phthisical patients show cerebral symptoms a few days before death, but at the autopsy no changes are found within the cranium. In these cases the diagnosis is not very important because the seriousness of the condition is evident from the other symptoms. The problem of the presence or absence of meningeal tuberculosis in phthisical patients has, however, a great prognostic value in cases showing a tendency to quiescence or cure, and the occurence of symptoms suggestive of tuberculous meningitis is more than disquieting.

In most cases the onset is insidious. For some days, at times for more than two weeks, the patient complains of headache. Tuberculous patients, even when they run fever, only rarely suffer from headache lasting more than a day or two, and then it is due to the pyrexia, or gastro-intestinal trouble. Acute rhino-pharyngeal inflammation may be the cause of headache, but this also is rare in tuberculous individuals. *If headache lasting several days, which cannot be explained as due to some evident cause, occurs in a phthisical patient, meningitis is to be thought of.* Vomiting is extremely rare in the early stages of tuberculous meningitis in adults. More common prodromal symptoms are irritability, fretfulness, confusion of ideas, impaired memory, photophobia, defective vision, drowsiness, and somnolence. In some, symptoms not unlike those of classical hysteria are observed for several days, and when occurring in a patient who had no such stigmata before, especially in males, hysterical manifestations are suggestive of meningeal irritation by tubercles.

The pulse-rate, which may have been accelerated, becomes slow in many, though not in all cases, and in some irregularity occurs. The temperature is high in some instances, but in most it hardly exceeds 102° F. I have seen instances in which the temperature was normal, or even subnormal. Constipation may occur even in those who had diarrhea. Another early symptom is retention of urine which, when it does occur, is of immense diagnostic value.

An important feature is a sudden change of character—the hopefulness and euphoria, which may have been more or less pronounced, change into indifference; the patient ceases to ask for relief, ceases to complain about the treatment given him, loses interest in his surroundings,

and lies in a semi-stuporous state unless waked, and then only answers questions abruptly.

In most of the cases under the writer's observation these symptoms occurred early, but it is important to mention that, as a rule, they were not continuous; occurring one day and disappearing the next, to reappear again. This intermittency is a very important point in the diagnosis of obscure cases. I have known cases in which the symptoms described above lasted about a week, then disappeared, the patient feeling quite well for one or two weeks, to relapse again when clear and unequivocal symptoms of tuberculous meningitis prove fatal.

During this stage some signs characteristic of this disease may be discovered. In most, Kernig's sign may be positive very early, though it is usually discovered later on. At times slight palsies, especially of the cranial nerves, may be noted when carefully examining the face. Transient monoplegia of an arm or leg occurs at times, in others, complete hemiplegia. Likewise aphasia occurs in some cases either as a transient phenomenon, or remaining to the end. Hyperesthesia, mainly of the muscles, is very common; so is dermographism. The pupils usually react to light and accommodation during the prodromal stage, but later on a defect in this regard is observed and there may be unilateral or bilateral ptosis, converging strabismus, and only rarely diverging strabismus. In some nystagmus occurs. An ophthalmoscopic examination may reveal tubercles in the choroid, which is conclusive as to the diagnosis.

Extensive experience has taught me not to rely implicitly on the results of lumbar puncture for diagnostic purposes in these cases. In most cases the fluid is found to come out at a high pressure, usually it is clear, in rare instances it is cloudy, rarely sanguineous, and often shows an excess of lymphocytes. But it must be mentioned that an excessive number of lymphocytes is not invariably a sign of tuberculous meningitis. It has been found in patients who did not develop the disease, while in many fatal cases with autopsy proof of tuberculous meningitis, no changes in the cerebrospinal fluid and its cytology could be found during life. Very frequently tubercle bacilli are found in the fluid. Excepting positive bacteriological findings, the writer would not make a diagnosis of tuberculous meningitis merely because of the high pressure, and the cytology of the cerebrospinal fluid alone, unless the symptoms are decidedly confirmatory.

Within a week or two after the onset of the prodromal symptoms the patient sinks into a stuporous condition, then into coma from which he can be waked with difficulty, and then the coma deepens until the end. Convulsions may occur, though this is comparatively rare. Retention of urine is very frequent during the last few days. A fatal prognosis should be given wherever meningitis is diagnosed; the few cases of recovery which have been reported[1] may be considered medical curiosities.

[1] See Harbitz: Am. Jour. Med. Sci., 1921, **161**, 212.

Diagnosis.—Acute miliary tuberculosis is not a disease which can be easily diagnosticated. When occurring in apparently healthy persons it is very often mistaken for some other acute infection, notably typhoid fever, septicemia, bronchitis, bronchopneumonia, malaria, etc. When attacking a patient who had been phthisical, the acute disease is at times considered by the attending physician as merely an acute exacerbation of the localized pulmonary lesion and a hopeful prognosis is rendered. In hospitals for advanced tuberculous patients the disease is more often suspected than it actually occurs.

It has been an ancient diagnostic principle to think of acute miliary tuberculosis in all cases of fever lasting more than a week and which cannot be diagnosticated as due to some other cause. In most cases it may be elicited that the onset was not as sudden as the patients claim, but that for weeks perhaps for more than a month, they have been feeling out of sorts, lost in weight, coughed mildly, perspired at night. In many, there will be found signs of a tuberculous lesion, active or latent, somewhere in the body; special inquiry should be made for symptoms and signs of tuberculosis of the pleura and the genito-urinary organs. In children the glands should be carefully examined.

The fever curve alone is not characteristic of the disease, though if watched for several days it may give some criterion. The lack of type in the fever, one day high, the next low, intermittent, or remittent, may help in excluding typhoid, pneumonia, malaria, etc., but the fever curve in septicemia often shows similar characteristics, while in acute miliary tuberculosis it may be continuous for a week, or more. Moreover, when complicating active pulmonary tuberculosis the fever curve may be the same as it had been before the onset of the complicating acute miliary tuberculosis. In senile patients the course may be altogether afebrile. Cornet observed such cases in young adults, and he concludes that it is a sign of cardiac weakness. The duration of the fever is at times misleading, because there are cases of miliary tuberculosis which last for several months, and only when cerebral symptoms make their appearance is the true condition recognized.

While with the meningeal implication the pulse may be slow, especially in children, it is rapid, soft and compressible in all other forms of the disease. The low blood-pressure may be of assistance at times. But the enlarged spleen, which is quite common, may lead one to suspect typhoid fever. Increase in the number of neutrophile polynuclears in the blood, diminution in the number of lymphocytes, and lack of eosinophiles, which are often observed, are of little diagnostic assistance in cases complicating chronic phthisis because this blood picture is common in cases of active phthisis without miliary complication. This blood picture is also often found in septicemia, and in pneumonia, though in the latter an absolute increase in the number of leukocytes is almost invariably observed. When, in an obscure case, there is observed an increase in the polynuclears, combined with leukopen[illegible]herculosis is to be considered likely. The

diazo-reaction is positive early, and persists throughout the disease, while in typhoid fever, it becomes positive only during the third or fourth week. Tubercle bacilli in the blood may be found in chronic as well as in acute miliary tuberculosis. However, a positive Widal reaction is rare in miliary tuberculosis. While in some cases tubercles may be seen in the choroid on careful and competent ophthalmoscopy, some authors even saying that this is true in 80 per cent of cases, this is a late clinical phenomenon, when other symptoms are decisive. Post mortem they are very commonly found.

After excluding typhoid fever, pneumonia, septicemia, etc., in a case of prolonged fever acute miliary tuberculosis is to be considered. When there is a rapid pulse, severe dyspnea, cyanosis, and a hacking, unproductive cough without any conclusive symptoms and signs of cardiac or pulmonary disease, the suspicion rests on a firmer foundation. In most cases it is the disharmony between the severe constitutional symptoms, with the negative findings in the chest, that leads to a diagnosis. In patients with pronounced tuberculous lung lesions, the sudden onset of higher fever, dyspnea, and cyanosis, when not due to pleurisy or pneumothorax, suggests complicating miliary tuberculosis.

Roentgenography.—When well developed, the lesions of acute miliary tuberculosis of the lungs are easily detected with the aid of a good roentgen plate. As was already stated, single tubercles cast no distinct shadows; only when several tubercles conglomerate in one spot that they produce a round or oval shadow which is more or less characteristic. These spots are seen scattered all over the pulmonary fields, more densely over the upper lobes, and at the roots of the lungs. Each spot represents a conglomeration of tubercles. In appearance they have been compared with a thick snowstorm. In some places these foci conglomerate more densely and produce an opacity on the plate which is much larger than the rest of the foci. In some cases the individual foci are not at all visible on the plate because the entire pulmonary field is opaque owing to the small size of each individual tubercle, and their larger number, and also because of the pulmonary edema which is a concomitant of this form of miliary tubercle. Cloudiness of the pulmonary field is the only thing that may be observed in these cases.

Though characteristic, these scattered foci should not decide in favor of miliary tuberculosis when the constitutional symptoms are not in agreement. Plates taken in cases of pneumokoniosis show pictures exactly like those of miliary tuberculosis, and the most expert is likely to be deceived. This has been a source of error which led astray many roentgenographers. It strikes me that the following points may assist in differentiation of such plates: In pneumokoniosis these foci are, as a rule, irregularly distributed all over the lung fields, especially in the region of the roots of the lungs where the glands retained the dust, and each spot differs materially in appearance and size from the others, while in miliary tubercle they are nearly all alike in size and appearance

and there is a tendency to segregation in the upper parts of the lungs. However, in advanced miliary tuberculosis the conglomeration of tubercles may also produce larger foci in some places on the plate. This again shows that only the constitutional symptoms decide.

Another difficulty in diagnosis arises at times. A patient with pneumokoniosis develops a tuberculous lesion which is localized and circumscribed, perhaps even fibroid in character, and the prognosis is quite favorable. A roentgenographic plate is made and disseminated foci all over the lung fields are observed in addition to the localized lesion in one of the apices and a diagnosis of miliary tuberculosis is made. In such cases a history and clinical observation for some time decides.

Miliary carcinomatosis, when hematogenous metastasis has taken place, produces a roentgen picture of the chest which cannot be differentiated from that of miliary tuberculosis. I have seen two cases in which this difficulty had arisen and it took some time before it could be cleared up. Usually the original neoplasm in some of the abdominal viscera, the thyroid, etc., can be found clinically, but now and then the diagnostic difficulties are perplexing.

Course.—The course of the disease is nearly always to a fatal termination. This may be expected within three or four weeks, but in rare instances it has been observed to last for several months. I have had cases in the hospital that lasted four and even six months, finally succumbing to meningeal implication. Even the meningeal form, in rare instances, lasts more than a month, with several remissions, apparently indicating that an error had been made in diagnosis, but within a few days, in one case after two weeks, the symptoms of meningeal irritation returned and the patient died. Cornet mentions instances in which the period of remission lasted over a month.

Patients presenting symptoms of the pulmonary form of the disease often go into the typhoid state and die in coma. Many show symptoms of edema of the lungs during the last few days, while convulsions are rare in adults. There have been reported cases of acute miliary tuberculosis which recovered, even such as showed tubercle bacilli in the cerebrospinal fluid, yet this is so extremely rare that it is not to be considered after the diagnosis has been positively made. Some last for months, as has been stated, and some authors have spoken of *chronic miliary tuberculosis*. In these cases several diagnoses have been made before the patient showed finally symptoms and signs of the typhoid, or meningeal, form of miliary tuberculosis.

CHAPTER XXIV.

PULMONARY TUBERCULOSIS IN CHILDREN.

General Characteristics of Tuberculosis in Children.—In children infection with tubercle bacilli, if it causes active disease at all, is usually followed by a generalized morbid process with implication of the lymphatic glands. This characteristic is the more accentuated the younger the child. In fact, in all infectious diseases we may note that the reaction of the lymphatic glands is intense in children. The glands are particularly sensitive to tuberculosis, and it appears that during infancy they are unable to retain the organisms, but permit them to spread all over the body.

The localized disease of the lungs peculiar to phthisis in adults, or in the bones and joints, characteristic of early childhood, is only rarely seen in infants. In infants tuberculosis is an acute, general infection, like typhoid or septicemia, and when the bacilli localize themselves by metastasis in any part, they produce lesions akin to those of pyemia.

Because of the implication of the glandular system, especially the intrathoracic glands, it was assumed by many authors that infection in children is invariably accomplished by inhalation of the bacilli. The microörganisms are deposited in the lungs, and when attempting to invade the blood, they are retained by the lymphatic glands.[1] When the localization of the lesion was found in the mesenteric glands, it was clear that ingestion of the bacilli was the channel of entry, and this was confirmed by the fact that in mesenteric tuberculosis bovine bacilli were often found.

But we have seen that this is not necessarily the case. Entering *via* the digestive tract, the bacilli may reach the tracheobronchial glands with as much ease as when entering *via* the respiratory tract. Behring and Calmette and their school maintain, in fact, that all tuberculosis, especially in children, is lymphogenous and hematogenous (see p. 54).

From the facts presented in the chapters on Phthisiogenesis it is clear that tuberculosis during infancy and childhood is hematogenous, irrespective of the portals of entry of the bacilli. A study of the rates of mortality during the various ages of life confirms this view. As will be seen from the accompanying diagram (Fig. 90), pulmonary tuberculosis is a frequent cause of death in infants under two years

[1] See Allen K. Krause: Studies in Tuberculous Infection, Am. Rev. Tubercul., 1919, **3**, 1.

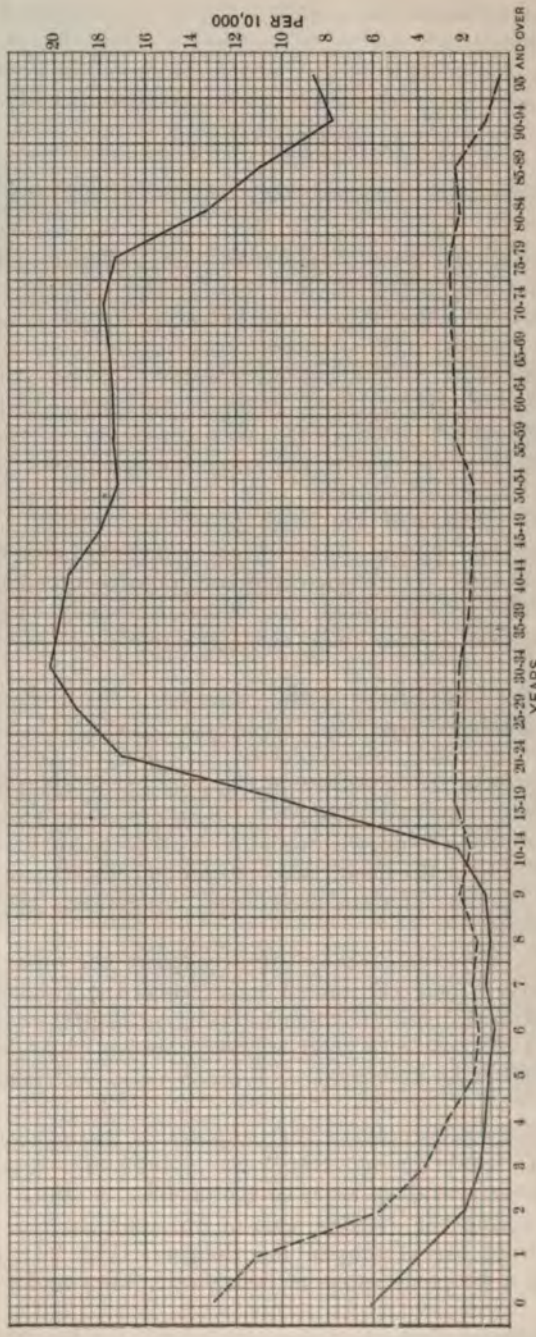


FIG. 90.—Mortality from tuberculosis per 10,000 living at the indicated age periods in the United States Registration Area, 1910 to 1913. Black line represents pulmonary tuberculosis, dotted line all other forms of tuberculosis.

of age; between three and fourteen years of age comparatively few succumb to this form of the disease; only after fifteen years of age does it become very frequent and remains so until the age groups above eighty years. We know from clinical experience that when occurring during the first two years of life, pulmonary tuberculosis is almost invariably acute, and the chronic type is extremely rare at this age. On the other hand, all other forms of tuberculosis, including that of the glands, bones, joints, serous cavities, especially the meninges, and the intestines; in short, the hematogenous forms of tuberculosis, cause death most frequently during the first four years of life and are comparatively uncommon as a cause of death after the fifth year of life.

It is thus clear that acute tuberculosis, as well as the hematogenous forms of this infection, have a different age incidence when compared with chronic phthisis, the disease which creates the main problem. Moreover, as was already shown, during the years when most of the human infections take place, between the second and the fourteenth, the mortality from all forms of tuberculosis is comparatively low; even hematogenous tuberculosis as a cause of death maintains the same rate throughout the rest of human life. It also shows that *phthisis, which is a common cause of death in adults, is not necessarily preceded by infection with tubercle bacilli immediately before the disease manifests itself by symptoms.* It shifts the problems of infection from the adult to the child.

TUBERCULOSIS DURING INFANCY.

We have shown that the child is born free from tuberculosis, and that infection, if it takes place at all, occurs post partum. Virchow, whose autopsy experience was as immense as that of any physician, stated that he never encountered a case of fetal tuberculosis. Infection in an infant is therefore invariably primary and almost always followed by symptoms of disease. Indeed, as we have already shown, there are cases on record in which infants brought into contact with a consumptive for an hour or so developed tuberculous disease of a malignant type. When the infection is massive, acute general tuberculosis with implication of the glandular system, and often of the lungs, is almost invariably caused.

The infant's organism behaves after a primary infection just as the very susceptible guinea-pig, the reason being that there is a primary infection of a body which has not yet been immunized by a previous mild infection. These cases occur mostly in infants who live with tuberculous persons—the father, mother, sister, brother, or nurse being tuberculous and, in handling the infant, an opportunity is afforded to transmit the disease. Thus, Combe[1] found a family history of tuberculosis in 90 per cent of his cases, if the word "family history" included

[1] Le Nourisson, 1916, **4**, 1.

all persons who live in intimate contact with the family. Clinicians have found that in doubtful cases a careful family history is a great aid in diagnosis, provided it includes not only the father and the mother, but also brothers, sisters, servants and relatives and acquaintances who come to the house, and in contact with the infant. There is evidence tending to show that in some cases, though in less than is generally supposed, the infection is derived from bovine bacilli through milk from tuberculous cows.

FIG. 91.—A primary cheesy focus the size of a lentil in a bronchus of the left lower lobe with miliary and conglomerate tubercles of the regional peripheral atelectatic lung. Caseation of the bronchopulmonary and lower tracheobronchial glands in the region of the right lower lobe. The glands on the left side are free. (Anton Ghon.)

In many cases no exciting cause, except the source of infection, can be traced. In others some acute endemic disease of infancy is found to have produced a state of allergy. This is especially true of measles and whooping-cough, but any of the other contagious diseases of infancy may reduce the vitality and resisting powers of the infant, and infection is then followed by the characteristic acute form of tuberculosis.

The period of incubation of tuberculosis in infants has not been exactly determined. In the few cases reported by Koch and Knipfelmacher[1] it appeared to be about seven weeks. Reuben[2] in New York found it to be from five to six weeks.

Symptoms.—The symptoms depend on the mode of onset and on the parts of the body which bear the brunt of the infection. In those in whom tuberculosis follows in the wake of another disease, like whooping-cough, measles, etc., there are usually symptoms of bronchopneumonia or meningitis, which carry off the patient within a few days or a week or two. In addition to the symptoms and signs of bronchopneumonia, there are often found enlargement of the spleen and liver, and swelling of the superficial glands, the cervical, axillary, inguinal, etc. This form of acute tuberculosis is best seen in cases of tuberculous disease engendered by inoculation, as in infection of the wound after ritual circumcision. Arluck and Wincouroff,[3] and Holt[4] have described such cases in detail.

In those in whom the disease is slower in development, athrepsia is seen. It is noted that the child does not thrive despite the fact that its nourishment leaves little or nothing to be desired and the gastrointestinal functions are fairly normal. There may be no fever at all. Still the emaciation proceeds frightfully. In some cases the emaciation consumes nearly all the subcutaneous adipose tissue and the thin, pale skin is stretched over the atrophied, softened, and bent bones. These infants usually have long hair on the back between the shoulderblades and on the extremities; their eyes are sunken and the eyelashes are frequently long. In a large proportion, over 20 per cent according with T. C. Hempelmann, tuberculides are found on the body. Finally the temperature begins to rise and may reach very high, and they succumb to symptoms of septicemia or meningitis.

Examination of the chest may not show any changes, while in some we may find areas of defective resonance, bronchial breathing, or rales. In infants limited and circumscribed lesions are very difficult of localization because we have no assistance on their part while exploring the chest.

Cough may be absent altogether, but in some cases we meet with a peculiar cough caused by pressure of enlarged glands on the bronchi, or on the nerves passing through the chest. Eustace Smith[5] first described this cough as spasmodic, occurring irregularly in paroxysms like those of pertussis, lasting only a short time and ending sometimes, though rarely, in a crowing inspiration. This cough has since been differently described by various authors. Schick[6] describes a respiratory crow, or stridor, resembling the sound heard in asthma and in

[1] Ztschr. f. Kinderheilk., 1915, **13**, 89.
[2] Arch. Pediat., 1916, **33**, 171.
[3] Beitr. z. Klin. d. Tuberkulose, 1912, **22**, 341.
[4] Jour. Am. Med. Assn., 1913, **61**, 99.
[5] Wasting Diseases of Infants and Children, London, 1878.
[6] Verhandl. d. Ges. f. Kinderheilk., 1909, **26**, 121.

capillary bronchitis. It can, however, be distinguished from the latter by the fact that in asthma the cough is paroxysmal while the stridor in bronchial adenopathy in infancy is continuous, lasting without change for weeks and months. The French have described it as *toux coqueluchoide*, and Striker compares it with the bark of a hoarse puppy.

In many cases *dyspnea* is observed. It may be inspiratory or expiratory, though in infants it is most commonly expiratory. It is best differentiated from dyspnea due to trouble in the larynx by the fact that in adenopathy the voice remains clear. With the growth of the glands, the dyspnea increases in intensity and in extreme instances asphyxia occurs as a result of occlusion of a bronchus, either suddenly or slowly. In these cases cyanosis of the extremities, face, etc., is marked, and inspiratory retraction of the lower parts of the chest, is a striking sign, etc. Many of these succumb to pulmonary edema.

In most of these slow cases the cachexia progresses until finally the child succumbs to some intercurrent disease or to tuberculous bronchopneumonia. In rare instances a softened gland ruptures into a bronchus causing aspiration pneumonia. A relatively large proportion end with tuberculous meningitis. Investigations made by the writer[1] of children under six years of age living in a tuberculous milieu in New York City have shown that 16 per cent succumb to meningitis, as against only 2.6 per cent among the general population.

Other infants may be anemic and underfed for months. They do not thrive in spite of all efforts to improve their nutrition. Finally, the marasmus assumes an acute character, the fever rises and they succumb to exhaustion or more commonly to some intercurrent disease.

Diagnosis.—It is clear that the diagnosis of tuberculosis in infancy is not an easy matter. Hamburger's[2] advice should be followed by all who have infants under their care: *Think of tuberculosis in every case in which no other diagnosis can be made.* This dictum is shared by nearly all other pediatrists who have given thought to the problem. Tubercle bacilli cannot be discovered because infants do not expectorate. Holt has, however, often found them by swabbing the throat with a pledget of cotton. A positive tuberculin (von Pirquet) reaction in an infant under one year is sufficient to clinch the diagnosis. Unfortunately during the course of measles or whooping-cough, and in tuberculous meningitis, the tuberculin reaction is apt to be negative, despite the presence of tuberculous infection.

Prognosis.—The prognosis of tuberculosis in infancy is very gloomy. In fact, it may be stated that *the younger the infant the more unfavorable the prognosis.* During the first three months of life hardly any survive infection; the vast majority of those infected during the second three months of life succumb to the disease or to some intercurrent infection; the outlook for infants between six and eighteen months is very unfavorable when infected with tuberculosis. Infants

[1] Arch. Pediat., 1914, **31**, 197.

[2] Brauer, Schröder, and Blumenfeld's Handbuch d. Tuberkulose, Leipzig, 1915, **5**, 6.

infected by tuberculous mothers succumb earlier than those infected by their fathers or others. Herbert Koch[1] suggests that the reason is probably that mothers infect their infants much earlier than fathers.

It appears to be the consensus of opinion of most pediatrists that all tubercles during the first two or three years of life are active, that the lungs show no tendency to limitation of the disease, and that there are no reparative processes to be noted when examining the lungs of infants who succumbed to tuberculosis. No cicatrization or calcification is to be observed.

The corollary has been drawn that all infants showing signs of infection with tubercle bacilli—a positive von Pirquet reaction—are doomed. The writer cannot agree with this. We have followed infants showing positive von Pirquet reactions during the first three months of life growing into healthy children. It appears that *the dangers of developing active tuberculous disease and the acuteness of the process engendered are in inverse ratio to the age at which the infection takes place. The younger the infant the more unfavorable the prognosis.* But even among very young infants cicatrization and calcification of the lesion may occur. In another place I have collected evidence showing that such healed lesions were found at autopsies made on infants who died from other causes. The writer has observed numerous infants living with their tuberculous parents become infected with tuberculosis, yet they grew into healthy children. Some have been followed for more than ten years. Mark S. Reuben in New York had under his care from 1909 to 1916, for shorter or longer periods, 23 infants who gave a positive tuberculin reaction. Nine of the 23 infants who became infected during their first year of life kept up in good health for from one to five years. T. C. Hempelmann studied the fate of 130 infants under two years of age with pulmonary tuberculosis. He found that the mortality among the infants under one year of age was 78.7 per cent; from one to two years of age, 57.4 per cent; and for the two years, 68 per cent.

It is thus clear that while tuberculous infection during infancy is very serious, it is by no means hopeless, as some writers have stated. At least one out of three survives.

TUBERCULOSIS DURING EARLY CHILDHOOD.

Significance of Tuberculosis during Childhood.—In our study of the epidemiology of tuberculosis we have seen that the child is born free from tuberculosis, but that sooner or later, coming into contact with tuberculous individuals, or their discharges, or consuming milk from tuberculous animals, it is infected with tubercle bacilli. We have also shown that during the first year of life relatively few—between 5 and 10 per cent—are infected with tubercle bacilli. During the

[1] Ergebn. d. inn. Med. und Kinderheilk., 1915, **14**, 99.

second year more are infected, and the number of infections keeps on growing so that at the age of fifteen over 90 per cent show unmistakable signs of harboring tubercle bacilli in the body. A study of the mortality from tuberculosis according to age groups has shown that the mortality from this disease is very high during the first two years of life. Considering the malignant clinical forms of the disease which have been described above, the reason is clear. But beginning with the third year the number that succumbs to this disease is very small and this low mortality keeps on until the fifteenth year, when there is another increase which keeps on rising, so that from the twentieth year onward the maximum has been reached, which keeps up until far-advanced age.

It is thus clear that *during the years when most infections with tubercle bacilli take place, the mortality is at its lowest.* It is also clear that if infection is to take place, which we have shown to be inevitable for those living in large industrial towns and coming into contact with many people, it is best that it should occur during childhood. Apparently, during this age period death due to tuberculosis is exceptional. This point will be discussed again when speaking of the prophylaxis of tuberculosis.

Infection and Morbidity.—We must again emphasize the difference between infection with tubercle bacilli, and disease due to this microorganism. It appears that *the vast majority of children infected with tubercle bacilli do not show any clinical manifestations of disease,* otherwise over 50 per cent of children in large cities would be sick and in need of careful treatment; at the age of ten over 75 per cent would be sick and in need of dietetic, specific, institutional, or climatic treatment. Scientific tests prove conclusively that the vast majority of children have been infected, and but few show clinical manifestations of disease; hence the bulk of infections at that age cause no disease, and may be disregarded by the clinician. Some, however, do show clinical manifestations of disease.

Tuberculous Adenopathy.—Scrofula.—A frequent manifestation of tuberculosis in children is enlargement of the glands, popularly known by the ancient and venerable name *scrofula.* Most commonly the cervical glands are affected, those below the angle of the lower jaw, or in front of the sternocleidomastoid muscle, and, in some, in the supraclavicular fossæ, become enlarged and hard. They may be so small as to be hardly palpable, but in others they become visible, and, in extreme cases, two chains of enlarged glands are seen on both sides of the neck, adherent to one another and to the skin. Later they may soften, rupture spontaneously, discharging pus, and leaving fistulæ which heal with difficulty, the remaining scars being ugly and disfiguring.

In some cases tuberculous lesions of bones develop, especially of the small bones of the carpus, tarsus, and phalanges; the epiphyses of the long bones are only rarely implicated. The common forms of these

osseous lesions are *spina ventosa* and *tuberculous dactylitis*, usually affecting the metacarpal bones and phalanges of the hands. The finger then is enlarged, bulbous, in characteristic fusiform shape, but slightly tender. In some instances the inflammatory induration is absorbed later, but more frequently softening and necrosis of the bone takes place, and sequestra are discharged, leaving ugly sinuses which heal sluggishly, and finally leaving more or less crippled fingers.

The tuberculous character of scrofulous lesions has been debated, but the consensus of opinion at present seems to favor the tubercle bacillus as the etiological agent. Cornet considers scrofula a product of mixed infection with pyogenic and tuberculous organisms, but the weight of recent opinion appears to be in favor of its being specific, and that no other organism outside the tubercle bacillus need be concerned in its etiology. Its relation to tuberculosis in later life has recently been discerned. Bartel and Eschrich have shown that

FIG. 92.—Multiple spina ventosa. (Tibler.)

scrofula is a form of infantile tuberculosis which occurs only in subjects possessing the lymphatic diathesis. In a recent study of the subject F. Spieler[1] arrives at the conclusion that the main channel of infection is the skin in which repeated inoculations of small numbers of tubercle bacilli takes place, and also the mucous membranes of the nose, throat and mouth. It is well known that children with the lymphatic constitution display strong tendencies to catarrhal conditions of the accessible mucous membranes. Scrofula may thus be looked upon as a manifestation of the defence reaction of the lymphatic child against a superimposed tuberculous infection. The lymphatic constitution is the preliminary condition in the clinical picture of scrofula; it is the primary, or predisposing cause. When in such a child tuberculous infection takes place, the glandular manifestations of the disease result.

[1] Skrofulose und Tuberkulose, Leipzig, 1920.

The relations of scrofula to pulmonary tuberculosis are rather interesting, and not appreciated to the extent they deserve. As will be shown later on (see Chapter XXX), it appears that scrofulous children only rarely develop pulmonary tuberculosis in later life, and when a lung lesion does occur, it runs a very mild course and tends to cicatrization, a fact which was already observed by clinicians several generations ago.

FIG. 93.—Tuberculosis of cervical and axillary lymph nodes in an eight-year-old boy. (Carr.)

On the whole, the prognosis of scrofula is very good, though the patient may ultimately remain with ugly and disfiguring scars on the neck. But these children are very susceptible to the acute endemic infectious diseases, and when attacked by them, especially by measles, whooping-cough, diphtheria, etc., the prognosis is serious. Escaping the ravages of these acute infections, they usually grow into healthy

men and women, and are less likely to succumb to pulmonary tuberculosis than others, as will be shown in another chapter of this book.

Not all enlarged glands observed in children are scrofulous, or tuberculous, in origin. Moreover, in *most of the children having enlarged glands, even of tuberculous origin, the symptoms are negligible, or there are no other clinical manifestations at all.* Thus, we often discover enlarged glands on the neck, or within the thorax, of children who are in excellent condition of health. In some we find the glands enlarged for some time, then there is a recession, the swellings go down or disappear, while the children had kept up their activities at school, and were none the worse for their experience. In others, the appearance of the glands is concurrent with the occurrence of some disease, like measles, scarlet fever, whooping-cough, etc.; they remain enlarged during convalescence, but after complete recovery they recede, or disappear permanently, or may return when some other exciting cause is again operative. We may thus see in many children a tendency to enlargement of the glands whenever an exciting cause is operative, but the innate forces of resistance are at work, and recovery takes place in a short time, spontaneously, or after some treatment has been instituted.

FIG. 94.—Tuberculosis of the submaxillary glands.

If we should take enlarged cervical glands as an invariable indication of active tuberculous disease in children, we would find very few raised under adverse economic and hygienic conditions who are free from this disease. Thus, among 692 children of tuberculous parentage examined by the author, 469, or 67.8 per cent, had swollen cervical glands. A careful examination of children attending dispensaries shows that between 50 and 75 per cent have palpable cervical glands. Most of them are due to carious teeth, hypertrophied tonsils and adenoids, stomatitis, eczema or pediculi of the scalp, etc. Correction of these conditions is usually effective in reducing the size of the glands. At any rate they are as a rule not tuberculous.

Symptoms of Glandular Tuberculosis in Children.—The appearance of glandular tuberculosis is accompanied, often preceded, by symptoms which are troublesome, and need careful study for their recognition.

Of these symptoms the following are the most important: Emaciation, fever, nightsweats, anemia, anorexia, etc.

Emaciation.—A healthy child gains in weight constantly, and if it is regularly weighed, say every month, it will be found that the scale registers more than at the preceding weighing. While in normal adults a lack in this direction is not necessarily an indication of disease, because they may have reached their normal standard, or even exceeded it, with children conditions are different. Commensurate with their gain in height, there must be a gain in weight in children of school age. It is known as the normal increment in the size of the body. When a child does not gain in weight it is, with few exceptions, an indication of disease.

To ascertain this gain in weight, various tables have been prepared by anthropometrists, and reproduced in many text-books on pediatrics. But I want to warn the practitioner against comparing the weight of a child under his care with that given in any of these tables. To begin with, the weight given in the table for each age is an average of a large number of children, and averages permit variations that are normal. The normal weight of the child depends solely on its height, and there are perfectly healthy children and adults who are short of stature.

What the physician should look for is a steady gain. If this is not found, it is clear that the child is sick. At any rate, it demands an explanation. In many cases it may be due to some intercurrent non-tuberculous disease. But it should be found and treated. *When we find that a child is not gaining in weight for several months, it is equivalent to a steady loss in an adult.* If there is no morbid condition to account for it, tuberculosis may safely be suspected as the cause. A careful physical examination will, in many cases, reveal enlarged intrathoracic glands.

An exception is to be mentioned. Infants may be suffering as a result of tuberculous infection and show no signs of emaciation for a long time. This is evident from the fact that tuberculous meningitis, or bronchopneumonia, often attacks well-nourished infants. Infantile tuberculosis, unless the gastro-intestinal tract is affected, does not always lead to cachexia.

With the emaciation there is often to be observed anemia, manifesting itself in marked pallor of the skin and mucous membranes, though an examination of the blood may not disclose any definite changes in its cytology.

Fever.—Whenever tuberculous glands cause trouble there is a rise in temperature. Hamburger's conception of tuberculous disease supplies the theoretical basis for the fever in these cases. He looks upon all clinical exacerbations of tuberculosis as spontaneous tuberculin reactions due to a sudden flooding of the body juices with tuberculin, producing the same symptoms as we produce artificially by injecting tuberculin. In other words, the fever is a manifestation of autoinoculation.

The healthy child's temperature oscillates between 98.8° and 99.8° F. Whenever it rises above these limits, it is to be considered patho-

logical, and an explanation is to be sought. If no cause can be found for elevation of temperature, which is observed persistently for several weeks, tuberculosis is to be thought of. In most cases it will be found that, in addition to the thermometrical findings, there are also symptoms of hyperthermia, such as anorexia, languor, etc. The child may feel refreshed and lively during the morning hours, but late in the afternoon it is flushed, tired, and seeks rest.

In evaluating thermometrical findings it must always be remembered that the *fluctuations in the temperature are much more pronounced in children than in adults.* Thus E. Wynn[1] found that among 1000 children 261 had temperatures of 99° F. or over, and of these, 112 presented no obvious pathological condition to account for the hyperthermia. Mary E. Williams[2] found among 1000 school children between the ages of twelve and fourteen years that no less than 55.5 per cent had temperatures of 99.6° F. and higher.

There are two reasons to account for the oscillations of the temperature in children. The heat regulating mechanism is more apt to be disturbed by slight factors than in adults, as is shown by the fact that nearly all pathological conditions produce higher fever in them than in adults. Then, there are so many subacute or chronic conditions which produce mild fever in children, that it would be wrong to base a diagnosis of tuberculosis on thermometrical findings alone. But when the temperature is found elevated persistently for several weeks in a child, and other symptoms of tuberculosis are present, while no other cause can be discovered, the patient is to be kept under careful observation. A difference of more than 1.5° F. between the minimum and maximum temperature of the day, when persistent, points to tuberculosis, when no other cause can be found.

Nightsweats.—As a symptom of tuberculosis in children nightsweats have not the same significance as in adults. *Many non-tuberculous children sweat during the night.* In a study of the physiological phenomena of sleep in children, Czerny[3] found that the intensity of evaporation from the skin goes hand-in-hand with the depth of the sleep. At the time when sleep is most intense, at its maximum, the skin is warm and moist, and usually profuse perspiration on the face is noted. This is not to be considered pathological.

To be of diagnostic significance, nightsweats in children must appear during the second half of the night and be so profuse as to soak through the bedclothes. Even in such cases they may not be pathognomonic of tuberculosis; the possibility must always be borne in mind that they may be of nervous origin, especially in older children. At any rate, nightsweats are often absent in tracheobronchial adenopathy, though with each exacerbation of the symptoms of activity they are to be observed.

[1] Public Health, 1913, **26**, 136.
[2] Lancet, 1912, **1**, 1192.
[3] Jahrb. f. Kinderheilk., 1892, **33**, 22.

In tuberculous bronchopneumonia in children nightsweats are the rule, but in non-tuberculous cases they are often a prominent and annoying symptom.

Cough.—Cough is another symptom of active tuberculosis in children. It is non-productive, unless the sputum is derived from the naso-

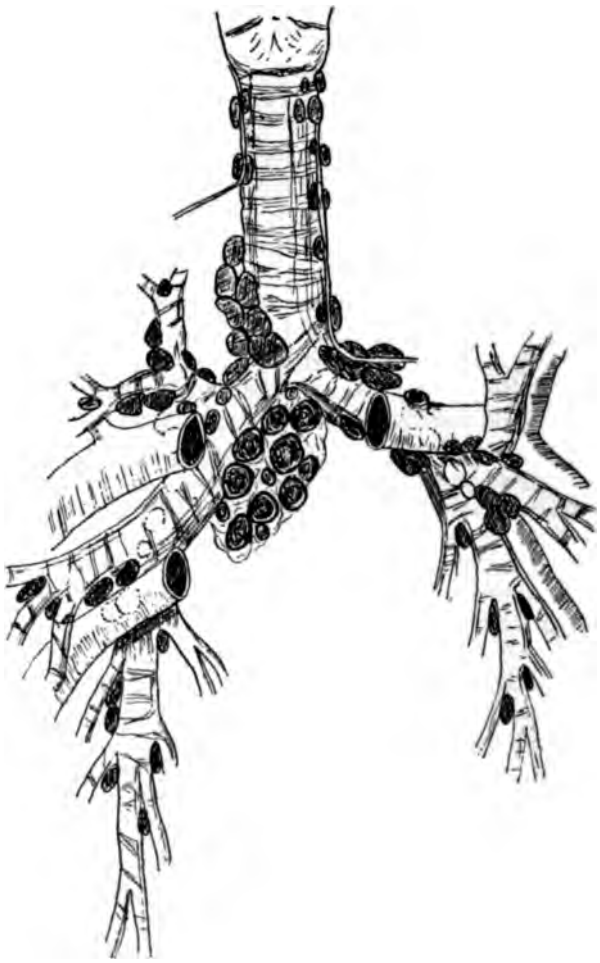


FIG. 95.—Diagram showing greater number of glands located on the right side.

pharynx, which is not uncommon. Hamburger says that it is never absent in active, incipient cases, and when cough lasts more than a week the possibility of tuberculosis should be considered and a thorough search for other symptoms and signs of the disease should be inaugurated. In advanced stages of the disease cough may be lacking, especially when there is an arrest in the progress of the disease, which is not

infrequently the case in children between eight and fourteen years of age. But even in these cases we meet with frequent exacerbations of the disease when the child coughs more or less.

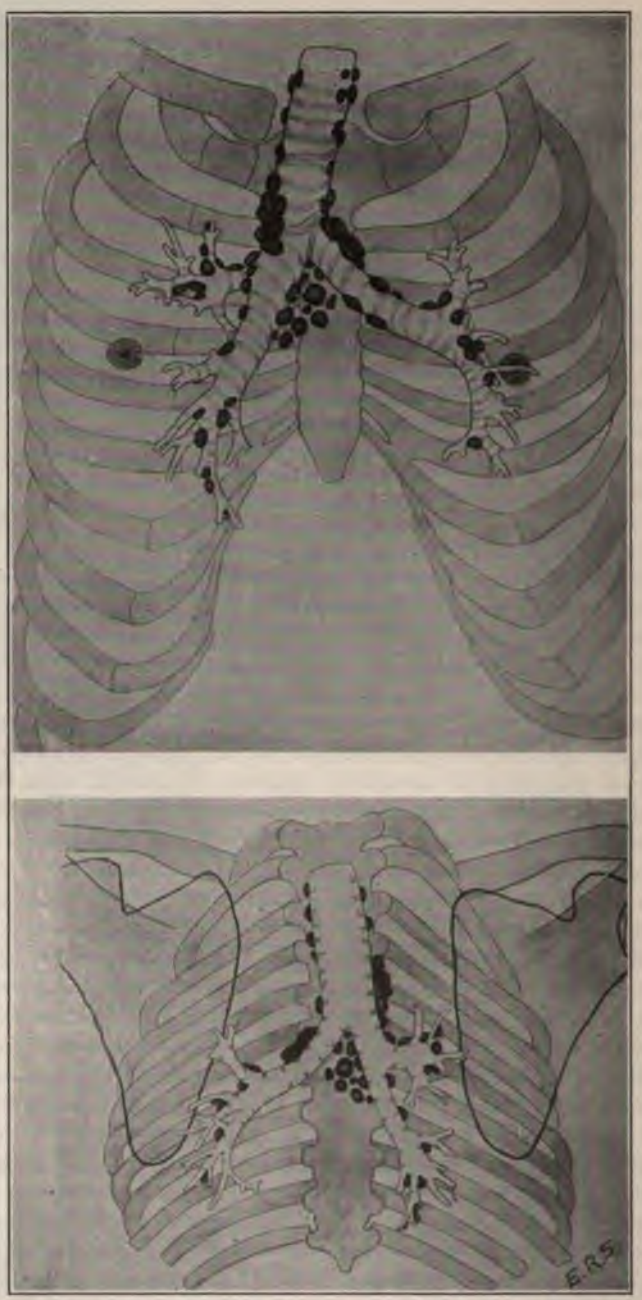
We must, however, emphasize that in children over three years of age cough is only of significance as a symptom of active tuberculosis when other symptoms are present, especially emaciation. When a child thrives, despite a chronic cough, it will be found that there is another cause, especially chronic or subacute catarrh of the nose and throat, particularly during the winter months. These children also may have enlarged cervical glands, which are neither tuberculous, nor scrofulous. In fact *tuberculosis is rare in children with hypertrophy of the lymphoid tissues of the rhinopharynx.* Asthma also is often a cause, and so is chronic bronchitis of the upper lobe, though we must be careful when finding unilateral bronchitis, which is almost invariably tuberculous. Signs of bronchitis of the lower lobe, even if unilateral, point to bronchiectasis and hardly ever to tuberculosis. Bronchiectasis is very common in children.

The paroxysmal and the brassy cough, as well as the expiratory stridor of infants, have already been described.

Children presenting any or all of these symptoms—emaciation, fever, nightsweats, cough, etc.—require a careful physical examination, and if these symptoms are due to active tuberculosis, we almost invariably find local tuberculous changes—that the glands are affected—except in those over eight years of age, among whom localized pulmonary tuberculosis of the same character as in adults may be found.

In many cases we note that despite the fact that the physical development of the child is decidedly inferior, its mental capacities are above the average. These children are often precocious, exceptionally good pupils at school, and if with artistic inclinations, they may be excellent musicians, etc. On the other hand, in quite a large proportion of cases the smouldering tuberculous process has quite the opposite effect—the child is backward in his studies, lazy and listless.

Physical Signs of Tracheobronchial Adenopathy.—The best that can be said about the physical diagnosis of tracheobronchial adenopathy is, that it is very indefinite; at any rate, all the criteria taken for proof of the existence of enlarged glands within the thoracic cavity do not enlighten us whether the process is active, and demands active treatment, or is merely an innocuous enlargement of the glands of no clinical importance, as it actually is in the vast majority of cases. Judging by the anatomical relations of these glands, it is clear that they must attain some size before they become discoverable by percussion and auscultation of the chest. But that they often do attain large dimensions may be assumed when we consider the size attained by the cervical glands at times. It may be said that there seems to be a relation between the size of the glands and the effects they produce. For this reason tracheobronchial adenopathy may be more easily diagnosed in infants in whom these glands are naturally large, than in children in whom they are comparatively small.



FIGS. 96 and 97.—Composite drawings showing the relationship of the bronchial glands to the thoracic wall in the adult. The glands are according to Sukiennikow, and the trachea and bronchi are after Blake (Am. Jour. Med. Sci., 1899, **117,** 320). In the child the trachea bifurcates at about the level of the intervertebral disk between the fourth and fifth thoracic vertebræ, which corresponds nearly to the tip of the fourth thoracic spine. This is about opposite the articulation of the third costal cartilage anteriorly. (Stoll.)

This group of glands includes those located around the trachea and bronchi, mainly in front of the bifurcation of the trachea. Pathologically, it has been found that those around the right bronchus are more liable to attain very large dimensions and produce symptoms and signs of the disease. From the practical standpoint, in addition to the anterior and posterior mediastinal glands, there are three groups which may become swollen because of tuberculous infection: At the bifurcation of the trachea we have the *tracheobronchial lymph nodes;* along the main bronchi there are the *bronchial lymph nodes;* and at the hilus of the lungs there are the *pulmonary lymph nodes,* which also surround the bronchi, and communicate through lymph spaces with the parenchyma. In fact, all these glands receive their lymph from the pulmonary tissue and the bronchi. Considering their anatomical relations it is clear that when enlarged, they may exert pressure upon the bronchi and trachea, as well as on the nerves and bloodvessels passing through the chest. They may produce symptoms because of pressure exerted on the vagus and recurrent laryngeal nerves and the superior vena cava. They may even press upon the phrenic nerve, the arch of the aorta, innominate veins, etc. But this is exceptional despite the fact that text-books give so many signs revealing pressure on the various structures. The anatomical relations of these glands are shown vividly in the illustrations (Figs. 96 and 97, page 462) from Stoll,[1] based on Sukiennikow's[2] anatomical researches.

Inspection.—On inspection the thorax is often found deformed in those who have had enlarged glands; indeed, some of the deformities produced by the intrathoracic glands are difficult to differentiate from the changes produced by early rickets. In some cases we find the typical phthisical thorax, the *habitus phthisicus*—a long, narrow chest with the ribs slanting downward at an acute angle, and narrow intercostal spaces. Children with such chests have passed through several attacks of glandular enlargement and may, at the time of examination, be in fair health. In many we see what Stoll calls the "hilus dimple." If the breath is held at the end of inspiration there is seen an apparent retraction on one or both sides in the second interspace. Owing to lack of expansion of one apex, the chest wall lags with inspiration. In old cases this "dimple" may remain permanently, owing to pleural adhesions or cicatrization of the peribronchial tissues at the hilus. (Figs. 98 and 99).

This phthisical chest, which some authors consider predisposing to phthisis, is in fact proof that the patient has been tuberculous for a long time, and in children it is proof that the thoracic glands have been enlarged. In our investigations of the form of the chest in children of tuberculous parentage, we found that at birth the chest is almost invariably normal, and only when tubercles affect the intra-

[1] Am. Jour. Med. Sci., 1911, **141,** 83; Ibid., 1914, **148,** 369; Am. Jour. Dis. Children, 1912, **4,** 333.

[2] Berl. klin. Wchnschr., 1905, **11,** 316, 347, 369.

thoracic viscera are changes in its form produced. In some cases unilateral bulging of the chest wall is noted, especially the first two interspaces near the sternum.

Enlarged veins are often visible on a chest containing enlarged glands. They are usually seen on the upper part of the thorax, mostly bilateral though not symmetrical, and at times unilateral. In my own cases, 37.5 per cent of children with tracheobronchial adenopathy had enlarged and visible veins on the thorax, and of these, three-fourths were unilateral. Of those in whom the diagnosis of latent tuberculosis was justified, or in whom it was strongly suspected, 25 per cent showed this sign, while among the manifestly healthy only about 1 per cent had enlarged veins on the thorax. Stoll found enlarged and visible veins on the thorax in 92 out of 173 cases; of these 50 per cent were tuberculous.

FIG. 98 FIG. 99

FIGS. 98 and 99.—The "hilus dimple." (Stoll.)

It thus appears that this is a fair sign of compression of the main trunks of the intrathoracic veins by enlarged glands or adherent pleura. My general experience, however, urges me against hasty diagnosis based on this sign alone. It is met with in many healthy children, especially such as have a delicate and transparent skin, and also in anemia. In adults, it is often seen in women during lactation, when it may be unilateral, and in persons suffering from non-tuberculous affections of the bronchi, lungs, and pleura, especially chronic bronchitis, asthma, and pulmonary emphysema (see p. 301).

Percussion.—A great deal has been written about percussion as an aid to the diagnosis of tracheobronchial adenopathy. But as a matter of fact there are many children with undoubted enlargement of these glands in whom the percussion note elicited over every part of the chest is practically normal. When we consider the topographical position of the bifurcation of the trachea, it is clear that the glands must become very large to produce dulness anteriorly or posteriorly over the surface of the chest. The various special methods like Koranyi's[1] vertebral percussion, which has been elaborated in this country by John C. Da Costa,[2] do not give satisfaction. In many cases, however, there is found paravertebral dulness on light percussion. The areas that may be found affected correspond to the hilus—the interscapular space, especially the right, and anteriorly in the upper two interspaces near the sternum. To elicit this, very light percussion is necessary because of the thinness and resilience of the thoracic walls in the child. It may be found when the glands are not very much enlarged; then it is due to the engorgement of the bloodvessels and lymphatics which exists in the region of the hilus during the course of certain acute infectious diseases. It is the collateral inflammation described by Tendeloo.[3]

This defective resonance is only rarely bilateral. Anteriorly it must be differentiated from the dulness due to an enlarged thymus. The latter is usually beneath the sternum, while in bronchial adenopathy the dulness is mainly at the side of that bone, mostly to the right. We must mention that there is normally an oval area of dulness between the first and fifth thoracic vertebræ, extending an inch or two outward on each side of the spine, to which William Ewart[4] has called attention. But in cases of glandular enlargement it is usually unilateral—one interscapular space is dull. I have seen a few cases in which enlarged thoracic glands produced dulness all over one side of the chest. It may be taken as an indication of compression of the main bronchus by the enlarged glands. Another point is that this dulness, to be indicative of adenopathy, must be permanent, found during several examinations. As has been pointed out by Grancher and J. E. H. Sawyer,[5] in debilitated and rachitic children there are observed transient areas of dulness, due to a bronchus being plugged with secretions and the resulting atelectasis of the air vesicles it supplies.

Auscultation.—In my experience auscultation has been of more service in attempting to diagnosticate intrathoracic glands. Normally the breath sounds in children are louder and somewhat harsher than in adults—*puerile*. But this, in healthy children, is heard all

[1] Ztschr. f. klin. Med., 1906, **60**, 295.
[2] Am. Jour. Med. Sci., 1909, **138**, 815; 1913, **146**, 660.
[3] Sixth Intern. Congr. on Tuberculosis, 1908, **6**, 197.
[4] British Med. Jour., 1912, **2**, 966.
[5] Birmingham Med. Review, 1912, **19**, 57.

over the chest. Swollen glands alter them in circumscribed areas. Thus, when large, we may find feeble breathing over a limited area, owing to compression of a bronchus, or to modifications in the pulmonary circulation in that region. In rare instances the breath sounds are feeble over a lobe, or an entire lung, anteriorly and posteriorly, which also is due to compression of the supplying bronchus. But this is liable to great variations. I have followed some children for years and found that at times there are modifications in the breath sounds in a given area which shift so that at the next examination, one or more months later, the modification is found at another place. It may be found that during an attack of an intercurrent disease, rhinopharyngitis, influenza, or any of the exanthemata, etc.—when the glands swell up and there is an exacerbation of the tuberculous process—the auscultatory phenomena make their appearance to disappear after the acute process is gone.

Anteriorly the auscultatory signs in children are uncertain, because normally we may hear the tracheal sound at the sides of the manubrium in emaciated, but non-tuberculous, children with narrow chests. Still, when tubular breathing is heard unilaterally at the side of the sternum it speaks for enlarged glands. Posteriorly, bronchial or harsh vesicular breathing in the interscapular space of one side is an indication of the transmission of the tracheal murmur by enlarged glands which act as sound conductors. In mild cases only prolonged expiration is heard in one interscapular space, but in those in which the glands are very much enlarged, the breathing over a limited area may be tubular, or exquisitely bronchial; almost the same as is audible when listening directly over the trachea.

D'Espine's Sign: Tracheophony.—About thirty years ago A. d'Espine[1] described a sign of enlarged tracheal glands which appears to be more satisfactory than any other symptom or sign at our command at present. It consists in auscultation of the voice, especially the whispered voice, along the course of the trachea posteriorly. He described this sign as follows: The patient is told to count "one, two, three," or "thirty-three," as clearly as possible (younger children may be told to say "papa," "mamma") while the examiner auscultates with the naked ear, or better with a stethoscope, the spines of the cervical vertebræ. So long as we listen to the cervical spines, we hear the characteristic tracheal tone and each word is quite clear. In a normal child this clear voice stops abruptly as soon as we reach the seventh cervical spine where the lung begins; but in cases with bronchial adenitis the clearness of the voice, or the tracheal tone, continues lower down, from the first to the fifth thoracic vertebra. It is at this spot that the main localization of the enlarged bronchial glands is found. The transmission of the tracheal tone in these cases is effected by the enlarged glands which surround the trachea at its bifurcation

[1] Traité des Malad. de l'enfance, Paris, 1900, p. 856.

and often reach the spinal column, acting as sound conductors between the trachea and spine.

When auscultation of the full voice gives uncertain results, the patient is told to whisper "thirty-three," which is even more reliable than the bronchophony just spoken of. It must always be borne in mind that in healthy children and adults, bronchophony and the whispered voice stop abruptly at the seventh cervical spine, and when heard lower it is a sure sign of something interposing between the trachea and the spine, and acting as a voice conductor.

This sign of tracheobronchial adenopathy has been extensively tried in France and many report that it is more reliable than any other sign. Barot[1] found it superior to percussion, and even more trustworthy than roentgenography for the purpose of ascertaining the presence or absence of enlarged thoracic glands. In this country it has been strongly recommended by Stoll, Sewall,[2] Howell,[3] Honeij,[4] and others.

In evaluating this sign it must be borne in mind that the height of the bifurcation of the trachea, where the glands are most likely to become enlarged in tuberculosis, differs according to the age of the patient. In infants and young children it is on a level with the seventh cervical vertebra. But with advancing age it sinks lower and lower. At the age of eight it reaches the second or third thoracic vertebra, and at twelve it is found as low as the fourth. In adults, especially in senile individuals, it may be found as low as the fifth or sixth thoracic vertebra. Therefore, in a child of ten, the transmission of the whispered voice to the third thoracic vertebra may not mean enlarged glands in the chest.

It must also be emphasized that the mere transmission of the vocal resonance as heard over normal lungs is not d'Espine's sign. This is found very often in children without enlarged glands. It is the transmission of the characteristic tracheal timbre which counts. In most cases it is heard not only along the spine, but also in the interscapular space on one side; at times bilaterally.

I have tested this sign in various ways and found it most satisfactory. In several cases the roentgenographic plate failed to disclose the presence of enlarged glands while d'Espine's sign revealed them.

Smith's Sign.—Eustace Smith's sign of bronchial adenopathy remains to be mentioned. It consists in this: If the child be made to bend back the head, so that the face becomes almost horizontal, and the eyes look straight upward at the ceiling above him, a venous hum, varying in intensity according to the size and position of the diseased glands, is heard with the stethoscope placed upon the upper bone of the sternum. As the chin is now slowly depressed, the hum becomes less loudly audible and ceases shortly before the head reaches

[1] Arch. médicales d'Angers, 1907, **12**, 18.
[2] Jour. Am. Med. Assn., 1913, **60**, 2027.
[3] Am. Jour. Dis. Children, 1915, **10**, 90.
[4] Jour. Am. Med. Assn., 1913, **57**, 958.

its ordinary position. Smith explains this phenomenon in this fashion: While the head is bending backward, the lower end of the trachea is tilted forward, carrying with it the glands lying in its bifurcation, and the left innominate vein, as it passes behind the first bone of the sternum, is compressed between the enlarged glands and the bone.

In my own experience this sign is not very reliable. It is found in short-necked children without enlarged glands, and is absent in many with adenopathy. Gibson[1] pointed out that it is mostly found in children who have enlarged veins in the neck and on the chest.

Reflex Symptoms.—There are other symptoms of tracheobronchial adenopathy which are described in great detail in text-books, but which are, in fact, very rare and may be left out of consideration in the average case. Thus, pressure on the recurrent nerve may produce paralysis of the right vocal cord; pressure on the sympathetic may produce differences in the size of the pupils. Pressure on the vagus may produce tachycardia and palpitation, transient or permanent. But these symptoms are very rare and are not conclusive even when encountered.

In younger children caseated glands may break through into adjoining structures, the bronchi, trachea, esophagus, etc. More rarely yet, the swollen glands acquire such dimensions that by pressure on a bronchus they prevent the entry of air into the part of the lung supplied by this tube; or by pressure on the trachea fatal asphyxia is produced. There have even been reported cases of rupture of a gland into the aorta. But these cases are extremely rare and may be considered medical curiosities.

Roentgenography.—With the enthusiasm of the first years of roentgenography, we thought that with the aid of the *x*-rays we had at last found a means for positively identifying enlarged tracheobronchial glands. Roentgenographers often made diagnoses of tuberculosis in children who showed no symptoms of active disease, and continued well indefinitely.

This was but natural, considering that normal glands allow the rays to pass through without casting any shadows, unless there is engorgement. Caseated glands cast a shadow which is occasionally distinct, but at times very indefinite. Only calcified glands cast a distinct shadow which may be identified in the vast majority of cases. But calcified glands, tuberculous in origin undoubtedly, are an indication that the disease has come to a standstill; in fact, this is the only mode of cure of caseated glands.

Under the circumstances the most easily diagnosticated cases of tracheobronchial adenitis, when the *x*-rays are used for the purpose, are those which have no significance clinically—those with calcified glands. When we attempt to clear up a case in which the glands are swollen, but neither caseated nor calcified—in other words, at a time

[1] British Med. Jour., 1906, **2**, 1051.

when therapeutic measures may be inaugurated with a good chance of helping the patient—the *x*-rays very frequently fail to give conclusive proofs of the existence of trouble. On the other hand, they show old and calcified glands which may not be, and often are not, the cause of the clinical symptoms for which the patient consults us at the time.

Fluoroscopy is of no value at all in most cases of young children who cannot be managed in a totally dark room, asked to breathe deeply, cough, etc. The best is a roentgenographic plate, taken instantaneously, and studied after it has been developed. But even here we must be careful before concluding that because there is a shadow at the hilus, there is active tuberculosis of the intrathoracic glands. In nearly all infectious diseases of childhood, especially in scarlet fever, measles and whooping-cough, these glands are enlarged, but the swelling slowly retrogresses during convalescence. In fact, de Mussy attributed the paroxysms of cough in pertussis to enlarged glands. It is therefore hazardous to diagnose tuberculous adenitis in a child with whooping-cough, or scarlet fever, as I have seen done.

Sluka[1] insists that several plates taken at long intervals are necessary, so that evanescent enlargements of the glands may be excluded. In fact, he found that the shadows shown on the plate of the same child at irregular intervals have been larger at one time and smaller at another; at times involving almost a complete lobe, or even a whole lung, at other times only a small circumscribed shadow was found; at one time in the right side, at other times in the left, etc. A considerable part of these changes is due to changes in the collateral inflammation in active cases, but it seems to me that differences in the technic of taking the picture, the distance of the tube from the patient's chest, the sharpness of the focus, the condition of the tube, etc., are responsible in many cases.

On the whole there is no doubt that shadows in the region of the hilus are indicative of enlargement or engorgement of the glands in that region. This mottling and stippling of the hilus is, however, no criterion as to the activity of the disease. Even the triangular or wedge-shaped shadow, with the base to the hilus, which has been described by Stoll and Heublein, Sluka, and others, is no proof of active disease, as the writer has repeatedly convinced himself. It appears also that in young infants these hilus shadows are only rarely seen even when adenopathy exists. Sluka says that in children under two years of age he never obtained a shadow on a chest plate which would even remotely suggest hilus tuberculosis, though he has taken numerous plates of sick children. He says that only during the third and fourth year do the glands begin to reveal themselves roentgenologically; they are mostly seen during the sixth and seventh years, and then begin to decrease in frequency.

Of late the confidence formerly placed in *x*-ray findings in intra-

[1] Wien. klin. Wchnschr., 1913, **26**, 254.

thoracic conditions has been waning. At the 1915 meeting of the American Pediatric Society,[1] Koplik said that "one should be very cautious in permitting an *x*-ray to make a diagnosis for him." Holt stated that he had "sent the same case to a roentgenologist on successive days and each day a different diagnosis was made. The *x*-ray is very misleading and a dubious procedure upon which to base a diagnosis."

In doubtful cases the roentgenographic plate may give some indefinite information about the presence of enlarged thoracic glands. But when found in a child showing no clinical symptoms of the disease, we must not conclude that the child is actively tuberculous. We do not as yet have enough experience with roentgenography in healthy children, nor have enough autopsies been made to verify roentgenographic findings, to warrant unequivocal conclusions.

Tuberculin Diagnosis.—Basing their opinion on the fact that tuberculosis in infants is almost invariably fatal, it has been concluded that when in a young child any of the tuberculin tests is positive, and there are some symptoms, such as cough, etc., the child should be pronounced tuberculous to the great dismay of the parents. I have seen children kept from school and thus deprived of an education, and perhaps hampered for the rest of their lives, solely because the von Pirquet reaction was found positive.

We have already shown that the tuberculin reaction indicates but one thing—whether the person—child or adult immaterial—has ever been infected with tubercle bacilli. But it does not show conclusively whether the infection was followed by disease. Inactive infection is more likely to give a strong reaction than active tuberculous disease. In fact, in fatal tuberculous bronchopneumonia, meningitis, influenza, etc., the reaction is negative; in others it is but faintly positive. In other words, the stronger the reaction, the less likelihood of active or dangerous disease in the child, and a negative reaction is no positive proof of the absence of dangerous tuberculous disease.

In infants under two years of age a positive reaction is to be taken as an indication of active disease because at that age infection is very likely to be followed by disease; during the first six months of life, almost invariably. But after two years of age harmless infections are the rule, so that the value of the tuberculin reaction acquires an academic importance, as was already shown, but it loses its clinical value. This is a point which pediatrists should bear in mind. It should never be lost sight of that after the third year latent tuberculosis is very common and this gives the same reaction as active disease.

Diagnosis.—The diagnosis of tuberculous tracheobronchial adenopathy depends on the presence or absence of clinical symptoms of disease. A child over two years of age showing a three-plus tuberculin reaction, and a shadow in the region of the hilus on the roentgenographic plate is to be considered well and healthy so long as it presents no

[1] Medical Record, 1915, **88**, 502.

symptoms of disease; so long as there is no fever, no cough, no emaciation, etc. It is different with those who have clinical symptoms. In these it is always important to remember that when a child does not thrive, fails to gain in weight, the cause must be found. If it is not found, and there is cough, especially the dry, brassy cough, the temperature is to be taken three or four times a day. If an irregular fever, of the type described above is discovered, there is presumption of tuberculosis. However, children with enlarged tonsils and adenoids may cough and have subfebrile temperature for weeks. But it must never be lost sight of the fact that these children only rarely develop active tuberculosis, as is shown when discussing the lymphatic diathesis in relation to tuberculosis (see p. 588). If on examining the chest we find some dulness in one of the interscapular spaces, or anteriorly in the upper two interspaces near the sternum; and the whispered voice and the tracheal tone along the spine and in one or both interscapular spaces are audible in the peculiar characteristic fashion described when speaking of d'Espine's sign, the diagnosis of tracheobronchial adenopathy is clinched.

It is different when these signs are found, even in conjunction with roentgenographic findings and a positive tuberculin reaction, in a child that shows no clinical symptoms of disease. There is no doubt that this child may have, and probably does have, enlarged bronchial glands. But these glands are not actively diseased, and so long as the little patient thrives, there is no cause for alarm. The glands are of no more clinical value than the scars found in the apices of 90 per cent of adults who die from causes other than tuberculosis; they are of no more serious import than the enlarged glands found on the necks of over 50 per cent of evidently healthy children in the slums of large cities.

Prognosis.—The prognosis of tuberculosis in children under ten years of age embraces two problems: (1) The immediate outlook; and (2) the ultimate outlook. In other words, what are the chances of survival, or of retaining good health, immediately after infection has taken place, and is the child destined to develop phthisis after reaching the age of adolescence?

The immediate outlook appears to be good, provided the lesions remain localized in the glands, or even in the bones and joints. This is clearly seen in cases of superficial glandular tuberculosis: Most children with tuberculous cervical adenitis, especially those requiring no operative interference, recover after a protracted illness. The same is true of osseous and articular tuberculosis. From 900 cases of tuberculous disease of the hip treated by A. Bowlby[1] at the Alexandra Hospital in London during twenty-one years, 33 died—a mortality of 4 per cent. He found that of the 33 who died, 24 were attacked by the disease before the age of six. The mortality from tuberculous tracheo-

[1] British Med. Jour., 1908, 1, 1465.

bronchial adenitis is undoubtedly even lower. The greatest danger is metastasis in the meninges, but even this is comparatively infrequent after the fifth year.

For this reason all methods of treatment of tuberculosis in children produce most excellent results. This is also the reason why orphan asylums—which harbor children between four and fourteen years of age—report that, despite the fact that most of their inmates are derived from the poorest strata of population, and an enormous proportion are of tuberculous stock, they have no morbidity nor mortality from tuberculosis. It is simply because death from tuberculous tracheobronchial adenopathy is extremely rare. The success of the open-air schools, the preventoriums, etc., should also be attributed in a great measure to this cause.

Barring meningeal complications, or intercurrent acute infectious diseases, the prognosis in tracheobronchial adenopathy is excellent.

In older children, seven years of age or more, the prognosis of apical pulmonary tuberculosis of the same type as seen in adults is more serious, though not so serious as in adults. It appears that pulmonary lesions in children heal with greater ease than in adults, though now and then we meet with a case in which the process in the lung proceeds to cavitation and the child succumbs to the usual clinical manifestation of phthisis. *After the twelfth year there is hardly any difference in the clinical picture and prognosis of phthisis in children and in adults.*

Says Franz Hamburger,[1] one of the most experienced men in this field: "In general we can lay down the fundamental principle that the prognosis of tuberculous manifestations in children is not at all bad. It is, in fact, one of the most important achievements of recent years that we know: 'Tuberculosis in children is a relatively harmless disease.' It will naturally take decades till the lay public will learn this important fact." And I may add till physicians in general will learn it.

The prognosis depends on several other factors: The younger the child showing active tuberculous manifestations, the worse the outlook, the more liable it is to suffer from, or to succumb to metastatic tuberculous manifestations, such as meningitis, rupture of a gland into a bronchus, the trachea, or esophagus. These complications in fact become less frequent after the third year of life, and after the sixth year they are comparatively rare. The prognosis also depends on various accidental complications. Thus, a child that escapes the endemic diseases, such as measles, whooping-cough, scarlet fever, diphtheria, etc., may grow up into healthy manhood, in spite of the enlarged glands in the chest which disappear leaving harmless calcified foci in nearly all cases after the tenth year; at any rate they give no more trouble. It is clear that the prognosis also depends on the

[1] In Brauer, Schröder and Blumenfeld, Handbuch der Tuberkulose, 1915, **5**, 31.

social and economic conditions under which the child is raised. Those who are well off in this regard have better chances to survive unscathed, because they have good nourishment, healthy dwellings, frequent vacations, and are less likely to contract other diseases, etc.

The second element in the prognosis of tuberculosis during childhood is the problem whether every child infected at an early age is destined to become phthisical after the fifteenth year of life. The facts observed in daily practice seem to be against such a view. If this were the case tuberculosis among adults would kill not only one out of seven to ten individuals, as is now found wherever there are available vital statistics, but over 90 per cent of humanity would succumb to phthisis. That an active tuberculous lesion during childhood is not necessarily followed by phthisis in later life is evident from the following facts:

We meet with many persons showing unmistakable signs of having had some form of tuberculosis during childhood, but pass through life as healthy and even vigorous individuals. This is the case with those showing scars on the neck which are undoubtedly remnants of tuberculous adenitis which had suppurated or was operated upon. We meet with many showing remnants of articular and osseous tuberculous disease, yet they pass through life without developing phthisis. In fact, the contrary seems to be true. Those who see large numbers of phthisical patients are struck by the fact that consumptives with scars on the neck, or with ankylosis of joints following earlier tuberculosis, etc., are extremely rare. This has been observed to be a fact by many clinicians, as is discussed elsewhere (see p. 584), and seems to indicate that an early tuberculous lesion may have some immunizing effect on the organism and prevent the development of phthisis in later life.

We are, at the present state of our knowledge, not warranted in asserting that this protection against phthisis conferred by early tuberculous disease depends on infection with bovine tubercle bacilli, as some have been inclined to assume. But we may safely draw a conclusion that *early tuberculous disease of the tracheobronchial glands is not necessarily followed by phthisis in later life, and there seems to be evidence that it may act in the same manner as articular, osseous, and glandular tuberculosis.*

CHAPTER XXV.

PHTHISIS IN THE AGED.

Frequency.—While discussing the frequency of tuberculosis during the various age periods we have shown that no age is exempt; in fact, it appears from available mortality statistics that after the age of twenty the death-rates from phthisis are about the same till very advanced life. While making autopsies pathologists are often struck with the frequency with which active tuberculous lesions are found in the lungs of aged persons, and investigations in homes for the aged show clearly that a large proportion suffer from phthisis. Thus, E. Braun[1] while making autopsies noted that in all bodies of persons over sixty years of age miliary tuberculosis was detected. The lungs were nearly always affected. In many the spleen, kidneys, and liver were involved; the meninges in only 10 per cent of cases.

The reason why popular opinion has ascribed immunity of old subjects to phthisis appears to lie in the fact that, when occurring, this disease runs a mild, benign course and may pass off as bronchitis, asthma, etc. But when the sputum expectorated by senile persons is examined, it is very frequently found to contain large numbers of tubercle bacilli. In fact, these aged consumptives may be considered actual bacillus "carriers" who, without themselves suffering very much from the bacilli, disseminate the disease much more widely than younger patients who know of their condition and the danger of indiscriminate expectoration.

Etiology.—Most phthisis in the aged has been acquired during childhood, but has been held in abeyance throughout life, to break out again at the period of life when the organs of the body begin to suffer as a result of wear and tear. Others have suffered from some form of phthisis before, but the disease was "cured," to reawaken during old age. Many have been afflicted for years with some form of fibroid phthisis, but when senile degeneration began to manifest itself the tuberculo-fibroid lesions in the lungs began to activate with more vigor.

From our present knowledge of phthisiogenesis we must exclude new infections of aged persons, because they have been infected during the earlier years of life, as was already discussed elsewhere. A new, or primary, infection in an adult would surely not pursue such a slow, sluggish course as is seen in the aged. The active disease in senile individuals should be considered either metastatic, or else old, perhaps dormant, processes flaring up and causing disease.

[1] Corr. Bl. f. Schweiz. Aerzte, 1917, **47**, 1121.

Pathologically, there are no differences in the lesions between the aged and those in adults in general, with but few exceptions. In the aged the fibroid processes predominate because the tendency to fibrosis of tissues is characteristic of advancing age. These fibroid formations tend to limit the lesion, prevent its spread and to surround the cavities, which show no tendency to enlarge by contiguity of the process. On the other hand, bronchiectatic cavities are more frequently found in older than in younger consumptives.

Symptoms.—"The conditions with which it may be associated modify the course of the tuberculous process," says J. Edward Squires,[1] "so that the symptoms are obscured and the signs of its presence in the lung are somewhat indistinct. Tuberculosis, when it attacks lungs already damaged by the degeneration of age, may add but little to the discomforts of the individual who is already short of breath and 'wheezy.' The increasing infirmity of the patient is accepted as a sign that he is ageing more rapidly, and no suspicion of any added mischief is aroused or entertained." Generally speaking, the symptoms of phthisis in the aged are often those of fibroid phthisis, which have already been described. From most patients who consult us for hemoptysis, cough, expectoration, and a lesion is discovered on physical examination, we elicit a history that they have been troubled with some of these symptoms for years, perhaps since childhood, but that they have been considered as suffering from chronic bronchitis or pulmonary emphysema.

The patients cough, but the cough is mild. In aged persons the stimulus for cough is not so intense as in the young because the sensibility of the bronchial mucous membrane is greatly diminished. The quantity of sputum they expectorate is, as a rule, not very considerable because they have a tendency to swallow it. When told that they are tuberculous they are apt to resent the imputation, claiming that they have coughed for years, perhaps since they can recall, and if it had been "consumption" they would have succumbed long ago.

Most senile patients are of slim build, but occasionally we meet with a tuberculous patient over sixty who is above the average weight. But with the onset of active symptoms they begin to lose in weight, and within a few months they may be reduced to mere skeletons.

A large proportion of patients have no fever, though the methodical use of the thermometer per rectum may reveal a typical tuberculous temperature with slight rises, to 101° F. in the afternoon. In this respect phthisis does not differ from other diseases in the aged. We know that pneumonia may pass an afebrile course in the senile. The organism of the aged does not react with fever as does the body of the young.

The pulse is more rapid than normal for the age of the patient. In rare cases tachycardia is seen, especially when there is cardiac dis-

[1] International Clinics, Sixteenth Series, 1906, **4**, 90.

placement. Dyspnea is a frequent symptom, especially after exertion. Because of the concomitant arteriosclerosis and myocarditis, cyanosis is not uncommon. In the later stages, when heart failure is apt to occur, edema of the extremities is frequently seen. The blood-pressure is low considering the age and the condition of the arteries of the patient. Hemoptysis occurs quite frequently. In most cases it is merely streaky sputum, but it may be profuse and I have seen a fatal hemorrhage in a woman, aged seventy-eight years. Nightsweats are rare because, with advancing age, the sweat glands undergo atrophy, and also because the great oscillations of temperature characteristic of phthisis in the young are absent in the senile.

A large proportion of aged tuberculous persons suffer from persistent diarrhea. In some it is very difficult to control by dietetic or medicinal treatment. Moreover, when the diarrhea is the dominant symptom, the symptoms and signs in the chest are overlooked, and a diagnosis of a gastro-intestinal disturbance is made. It is advisable that in all cases of persistent diarrhea in senile patients the chest should be carefully examined, and inquiry made about the constitutional symptoms of phthisis.

Physical Signs.—The appearance of the senile phthisical chest depends on the character of the lesions in the lungs. In those in whom there is pulmonary emphysema in addition to the tuberculous process there is the characteristic barrel-shaped chest, rigid, hardly expanding; in fact, always in the position of maximum inspiration. All that is seen is that the entire chest is lifted up with each inspiration, but there is no antero-posterior or lateral expansion. The intercostal spaces are wide and the direction of the ribs is more horizontal than normal. But many have no old emphysema and in them the thorax is rigid owing to the ossification of the costal cartilages; the ribs run at a more acute angle to the spine than normal and the intercostal spaces are wider; the supraclavicular and infraclavicular spaces are deeply excavated, more so on one side. During fits of coughing either apex, or both, may be seen blowing up in the supraclavicular space. Dilatation of the veins of the neck is a frequent symptom, and when there is relative tricuspid insufficiency, owing to dilatation of the right heart, there may be a venous pulse. Kyphosis and kyphoscoliosis are never absent.

Auscultation is also not so satisfactory as in young subjects. The breathing is superficial and, combined with pulmonary emphysema, which is only rarely absent, we may hardly hear any breath sounds, or only a feeble murmur is audible. These are also the reasons why bronchial or cavernous breathing is so rarely heard over the sites of cavities. Bronchovesicular breathing of low pitch, with prolonged expiration may, however, be made out over one apex, at times, while carefully auscultating the chest. Rales are not audible in many cases because of the superficial breathing; but over the sites of excavations large consonating rales may be heard, even when no breath sounds

are made out. At the base, these rales are usually due to bronchitis or bronchiectasis which are very frequent in old age.

Course.—In many cases the cough, expectoration, emaciation, etc., continue for years and, inasmuch as these old persons do not follow occupations necessitating physical exertion, the true nature of the disease is not even suspected. They are considered patients suffering from chronic bronchitis or emphysema. I know old consumptives who have survived children and grandchildren whom they infected with tuberculosis. In fact, whenever I discover children with signs of tuberculous infection, though a history of exposure cannot be made out, I inquire for the grandparents, and have, in many instances, found that one of them was the source of infection, though he did not know the true nature of his illness.

In the vast majority of cases the tendency of the disease is to progress, though slowly, and never to a cure. Occasionally we find that it advances rapidly, assuming an acute or subacute course, with hectic fever, rapid emaciation, etc. Owing to the weakness and the general debility the cough is usually not at all severe, and when there is no fever, a diagnosis of carcinomatosis is made. Others cough and expectorate for years, when suddenly fever develops and the patient is carried off within a few days. Bronchopneumonia may have been erroneously considered the cause of death, unless the sputum was examined and tubercle bacilli were found; a diagnosis of acute primary tuberculosis may then be erroneously made. Daremberg speaks of acute phthisis in the aged, and Hoppe-Seyler speaks of acute miliary tuberculosis on rare occasions. But these cases are evidently acute exacerbations of chronic phthisis which had been kept in abeyance for years. The large proportion of cases of acute miliary tuberculosis found by Braun while making autopsies on aged persons show that it is frequently overlooked by clinicians. He points out that paradoxical bronchitis and bronchopneumonia, with signs of heart failure, doubtless usually conceal the presence of acute miliary tuberculosis in the aged.

Diagnosis.—The diagnosis is not difficult when the possibility of phthisis is kept in mind in all cases of cough, expectoration, emaciation, etc., met with in senile patients. Most of the mistakes made in these cases are due to failure to examine the sputum for tubercle bacilli. When the physical signs in the chest are indefinite, which is often the case, the bacteriological findings decide. When looking for fever in these cases we should never rely on the axillary temperature; only the rectal is to be taken.

We must guard against mistaking signs of old, healed lesions for active disease. This can be avoided by a careful study of the symptomatology and bacteriology of the affection.

CHAPTER XXVI.

TUBERCULOSIS OF THE PLEURA.

THE serous membranes of the body, the meninges, the peritoneum, the pericardium, and the serous linings of the joints, are very much predisposed to tuberculous disease. As a serous membrane, the pleura is no exception in this respect. Indeed, it may be stated that tuberculosis of the pleura is at least as common as tuberculosis of the lungs. In all forms of phthisis the morbid process extends from the pulmonary parenchyma to the visceral pleura. Its anatomical relations, blood supply, and lymphatics, render the pleura peculiarly liable to infection with tubercle bacilli which, as we have already shown, spread within the body either hematogenously or lymphogenously. Of the two sheets, the visceral, especially the part covering the pericardium and diaphragm, is very thin and firmly adherent, while the parts covering the surface of the lungs are thinnest and detached only with difficulty. The costal pleura is thicker and covered with flattened epithelial cells, while the cells covering the visceral pleura are less distinctly flattened, more granular and polyhedral. The pleura rests on a thin layer of subserous areolar tissue containing numerous elastic fibers. These areolar and elastic fibers are continuous with the elastic fibers and connective tissue within the lungs.

The blood supply of the pleura is not an independent system, but is derived from two sources: The visceral pleura is, through its circulation, intimately connected with the lung, being supplied with branches of the pulmonary and bronchial arteries, but the capillaries beneath the visceral pleura form a coarser network than those of the pulmonary alveoli. On the other hand, the parietal pleura is supplied from the intercostal, phrenic, internal mammary, mediastinal and bronchial arteries. It is thus clear that disturbances in the bronchial and alveolar circulation may affect the pleura, especially the visceral sheet. Moreover, inflammatory conditions of the lungs, when extending to the surface, will almost invariably implicate the visceral pleura, while the parietal sheet will only be affected through contact.

The visceral pleura is very rich in lymphatic vessels and glands which are often visible to the naked eye. They are scattered all over the surface of the pleura, but are most numerous on the membrane covering the interlobar fissures. Their connection with the bronchial glands is evidenced by the fact that they too became darker in older individuals owing to the deposition of carbon particles brought into the lungs with the inhaled air. The lymphatics of the parietal pleura pass to small intercostal glands situated near the heads of the ribs, and

indirectly, through their connection with the lymphatics of the fourth and fifth intercostal spaces, with the axillary glands. There are also communications between the lymphatic systems of the chest and the abdomen through anastomosis between the lymph vessels of the pleura and those of the peritoneum, particularly that covering the lower surface of the diaphragm. At first sight a closed cavity, the pleura is thus seen to communicate through its bloodvessels and lymphatics, in the latter by means of stomata, with the air inhaled into the lungs as well as with the abdomen. Infection of any part of the lungs or its glands, or of the peritoneum, is likely to spread hematogenously or lymphogenously to the pleura. In fact, experimental investigations of Grawitz, Grober, Fleiner, and others, have shown conclusively that coloring matter insufflated into the lungs of animals was subsequently found in the pleura. Tubercle bacilli carried by the blood, or especially the lymph stream, may thus produce pleurisy even if the lungs remain unaffected. Primary tuberculous pleurisy is thus explained.

The lymphatic connections of the pleura have recently been studied in this country by William Snow Miller.[1] He found that the lymphatics of the pulmonary parenchyma drain directly into the bronchial lymph glands. But the pleura has a set of lymphatics entirely distinct from those of the lung substance proper. In the injected specimen they appear as a network of rather large lymphatic vessels, in the meshes of which are numerous smaller vessels. This pleural lymphatic system has an independent drainage of its own directly into glands at the hilus, except for the diaphragmatic pleura of the lower lobes which drains into the preaortic nodes *via* lymph vessels in the ligamentum pulmonale. The valves of the lymphatics leading from the pleura point toward the hilus and prevent the flow of lymph in the reverse direction. It is important to note that there is a connection between the lymphatic systems of the pulmonary parenchyma and the pleura by means of short lymphatic trunks which follow the branches of the pulmonary veins that take their origin from the pleura. The valves in these communicating lymphatics all point toward the pleura, so that the lymph in these vessels must always flow from the lung tissue toward the pleura, and not *vice versa*. The bearings of facts with respect to the lymphogenous transmission of tubercle bacilli, or other organisms, or even cancer cells, are obvious.

Pathology.—While making autopsies on tuberculous bodies we almost invariably find that the pleural sheets, in part, or even completely, are covered with fibrinous exudate, a false membrane; are adherent at some circumscribed area, or more extensively, and thickened.

An exudate in which fibrin filaments are more or less abundant is frequently found within the pleural cavity. In nearly all cases of chronic phthisis the pleural sheets over the affected upper lobe are thick, and densely adherent, so that the lung cannot be removed

[1] Am. Rev. Tuberc., 1919, **3**, 33.

without force; either the parenchyma, or the tissues on the inner side of the chest wall, must be forcibly torn or cut for the purpose. Next in frequency, thickening and adhesions of the interlobar and diaphrag-

FIG. 100.—Tuberculous pleural adhesions. At the lower part of the drawing is to be noted that the subcostal cellular tissue is very much reduced in quantity. Above it the new membrane is developed at the expense of the visceral pleura and shows a layer of tuberculous follicles. The fibrous tissue gradually extending upward and coming in contact with the lung without any sharp line of demarcation between them, is already old, well organized in parallel bundles and passed by numerous bloodvessels. (Chantemesse and Courcoux.)

matic pleuræ are found. The interlobar fissure near the affected part is thus often obliterated. In acute cases miliary tubercles may be found scattered all over the surface of the pleura; in others tuberculous neoformations occur; they may form large villous tumors which, in

rare instances, are found pedunculated as in bovine tuberculosis of the pleura. Very often calcified areas are made out in the affected part of the pleura which, at times, may be over one centimeter in thickness, and converted into a fibrous or even calcified mass which surrounds the diseased and excavated part of the lung like a solid shell.

Microscopically the false membrane in mild cases is made up of fibrin enmeshing red blood corpuscles and round cells. The pleura proper is invaded by young connective-tissue cells, tuberculous granulations, epithelioid and giant cells, and areas of caseation. The adjacent parenchyma of the lung usually shows atelectasis of the alveoli, vascular dilatation and proliferation of the epithelial cells.

In a certain sense, the implication of the pleura in pulmonary tuberculosis may be regarded as a protective process. The acute symptoms of pleurisy, especially the pain in the chest, impede the motion of the affected part of the thorax and thus afford rest to the diseased lung, favoring cicatrization of the lesion. But this is of less significance when compared with the protection pleural adhesions afford the patient against loss of continuity of the visceral pleura resulting in pneumothorax. When the tuberculous process reaches the cortical surface of the lungs, which it does in nearly all active and progressive cases, a minute caseated area will permit the entry of air into the pleura and cause collapse of the lung. The pleural adhesions over the site of the lung lesion prevent this accident in over 95 per cent of cases of phthisis.

Varieties of Tuberculous Pleurisy.—The following forms of tuberculous pleurisy may be differentiated clinically and pathologically:

1. *Primary tuberculosis of the pleura*, which is rare.
2. *Pleurisy during the course of acute pulmonary tuberculosis.* Met with in nearly all cases.
3. *Pleurisy during the course of chronic pulmonary tuberculosis*, encountered in various degrees of intensity and extent in nearly all cases of chronic phthisis.

Each of these forms of pleurisy may be dry or moist. The latter class may have serous, serofibrinous, sanguineous, or purulent effusions. It may be unilateral or bilateral; may involve the entire surface of the affected pleura, or only a limited area.

PRIMARY TUBERCULOSIS OF THE PLEURA.

Primary tuberculosis of the pleura is rare, if it occurs at all. It is clear that in such cases the virus must be brought to the pleura through the blood or lymph stream. Experimental investigation has shown that even when the pleura is directly inoculated in a healthy animal no local tuberculous lesion is produced. Cleveland Floyd[1] found that only when the pleura is sensitized by a previous infection for some days the response to infection with pyogenic microörganisms was in the

[1] Tr. Am. Climatol. Assn., 1914, **30**, 205.

nature of purulent effusion. Similarly, Robert C. Paterson[1] found that fluid is never produced by a primary inoculation of the pleura with tubercle bacilli. But in *tuberculous* animals inoculation of tubercle bacilli produces an exudate of serum, leukocytes, red blood corpuscles, and fibrin. He therefore arrives at the conclusion that clinical pleural effusions are caused by infection of an "allergic" pleura; in other words, that they are due to reinfections from within, or from without the body. This is confirmed by the clinical observation that pleural effusions are almost invariably preceded by many months, or years, by tuberculosis of some other organ in the body, notably the lung, the lesion remaining dormant. Pathologically also there are confirmations—in nearly all cases of tuberculous pleurisy that come to autopsy older lesions are found in the lungs, or the intrathoracic glands.

Tuberculous pleurisy is found more frequently in men than in women. While no age is exempt, it is mostly found in adults. Pleurisy in children, with or without effusion, is, as a rule, non-tuberculous. Many patients give a history of exposure to cold as the immediate exciting cause. When we bear in mind that it is an endogenous reinfection with tubercle bacilli, we can readily conceive that exposure to cold may prepare a suitable soil for the tubercle bacilli brought there by the blood, or by contiguity to adjacent diseased organs. The blood and lymph supply of the parietal pleura, being derived from that of the chest wall (see p. 478) will predispose it to inflammation after chilling of the chest wall. It may be stated, however, that the vast majority of these cases of "idiopathic" pleurisy are tuberculous. Autopsies made on persons with dry pleurisy, apparently due to "colds," have shown distinct tuberculous lesions of the lung and pleura. At times an injury is responsible for the onset of pleurisy. But it appears that individuals who do not harbor tubercle bacilli, or are otherwise not predisposed to tuberculosis, do not develop tuberculosis of the pleura after an injury to the chest. During the World War tuberculosis of the pleura was noted to follow injuries and wounds of the pleura only in exceedingly rare instances.

Symptoms of Dry Pleurisy.—In general practice dry pleurisy is very frequently observed. After exposure, or without any known provocative cause, the patient is seized with some chilly sensations, though the acute chill characteristic of pneumonia is very rare, becomes feverish, has pain in the side of the chest, and more or less dyspnea. Unproductive cough is almost invariably present, and it aggravates the dyspnea and the pain. In some instances paroxysmal attacks of cough occur which are very painful. Physical examination of the chest shows diminution of mobility, at times almost complete immobility, of the affected side of the chest. Percussion yields negative results, but auscultation reveals a dry friction sound, most commonly in the region where the pain is acutely felt—the lower part of the chest in the

[1] Am. Rev. Tuberc., 1917, **1**, 353.

anterior axillary line, or the mammary region, or behind, in the region of the angle of the scapula. In some cases the pain is mild, but in others it is severe, lancinating. It may be relieved, more or less, by anything which tends to immobilize the affected side of the chest, and is aggravated by deep breathing, or coughing.

As has been pointed out by Capps, the pain in pleurisy is only felt superficially; it is "referred," and can be elicited only in the skin, subcutaneous tissue, and muscles. The sensitized area is hyperesthetic, hyperalgesic, and often characterized by painful tender points. The muscular cutaneous reflexes are exaggerated, and can best be elicited by striking or pinching the skin. In most cases it is felt in the region of the affected pleura, *i. e.*, where the friction sound is heard; tenderness of the intercostal spaces may be elicited.

In *diaphragmatic pleurisy* no friction sounds are heard on auscultation and the diagnosis is made mainly by a consideration of the general and local symptoms. The fever is, in most cases, high and the dyspnea severe owing to the immobility of the affected half of the diaphragm, the result of the pain which may be agonizing. It is aggravated by cough, swallowing and abdominal breathing. For this reason the patient makes a strong effort to breathe mostly with the upper part of his chest, thus keeping his diaphragm at rest as far as possible. The diaphragm derives its sensory nerve supply from two sources—the phrenic, and the last six intercostal nerves. The central portion of the diaphragmatic pleura is innervated by the phrenic nerve. Hence, inflammation of the central portion sets up pain in the neck, at the crest of the shoulder, corresponding to the cutaneous distribution of the fourth cervical nerve, which has its center in the spinal cord at the same level as the phrenic. The periphery of the diaphragmatic pleura is innervated by the sensory fibers of the intercostal nerves and inflammation of that area gives rise to referred pain in the lower thorax, the lumbar region, or the abdomen. These points of tenderness in pleurisy were first studied by Gueneau de Mussy,[1] who described *boutons diaphragmatiques*, points of maximum tenderness at the intersection of the parasternal line and a horizontal line continuous with the end of the tenth rib. More recently Sir James Mackenzie,[2] and especially Joseph A. Capps,[3] have carefully studied the subject.

In many cases of diaphragmatic pleurisy the referred pain over the abdomen and back (Figs. 101 and 102) is not unlike that due to appendicitis, gastric ulcer, cholelithiasis, and other intra-abdominal diseases. Capps mentions cases in which such errors have been committed; Lewis Sayre Mace reports several in which gastric ulcer was diagnosticated, and I have seen many cases of this type, especially in tuberculous patients who have had recurrent attacks of diaphragmatic pleurisy and resulting adhesions.

[1] Arch. gén. de méd., 1853, **2**, 271; 1879, **2**, 141.
[2] Symptoms and Their Interpretation, London, 1910.
[3] Arch. Int. Med., 1911, **8**, 717; Am. Jour. Med. Sci., 1916, **151**, 333.

T. H. Kelly and H. B. Weiss[1] report a series of cases of diaphragmatic pleurisy which simulated surgical conditions so closely that the ques-

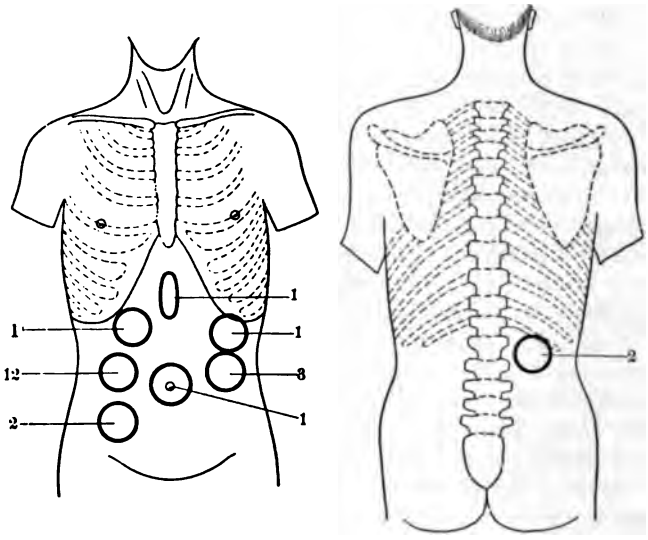


FIG. 101.—Points of maximum pain and tenderness in abdomen and back occurring in 61 cases of diaphragmatic pleurisy. (Capps.)

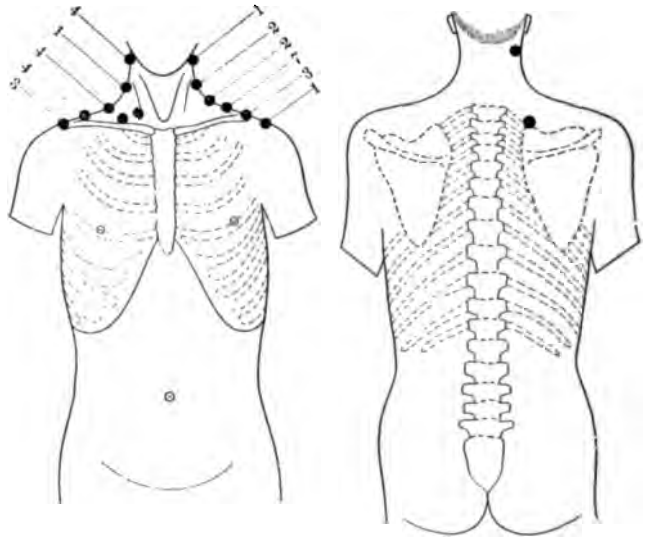


FIG. 102.—Points of maximum pain and tenderness in the neck region occurring in 61 cases of diaphragmatic pleurisy. (Capps.)

[1] Am. Jour. Med. Sci., 1918, **156**, 808.

tion of operative intervention was seriously considered. Among the diseases which required differentiation were renal stone, acute cholecystitis, generalized acute peritonitis from perforated typhoid ulcer, etc. Some had in fact been operated upon for appendicitis and gall-bladder disease previous to coming under Dr. Kelly's observation. In none of those operated cases was stone or any other pathological condition of the intra-abdominal viscera found at the operation, and shortly afterward there was a recurrence of the symptoms that had existed before the operation.

Several cases of diaphragmatic pleurisy have come under my observation in which the diagnosis was made of chronic recurrent appendicitis, and operated upon; others in which operation was performed for gastric ulcer because of hemorrhages which in reality were due to tuberculous lesions in the lung, or bronchiectasis which is not uncommon in chronic diaphragmatic pleurisy. One patient with a thick pleura over the right base was operated upon four times: For appendicitis, for gall-stones, for gastric ulcer, and finally for "adhesions." When he came under my observation he still had pain in the right side of the abdomen, and a surgeon urged another operation. We have already shown that hematemesis is at times difficult of differentiation from pulmonary hemorrhage (see p. 251). In tuberculous patients, symptoms of appendicitis, especially of the chronic and recurrent type, should be carefully analyzed before a final opinion is given.

The differentiation may be attempted along the following lines: In diaphragmatic pleurisy there are two areas of tenderness on pressure: One posteriorly along the twelfth rib of the affected side; the second at the ridge of the trapezius. Spontaneous pain at these points may occur, but pressure elicits it in nearly every case. In some instances there is observed pain and rigidity of the abdominal muscles of the affected side. When due to intra-abdominal disease deep pressure will produce severe and deep-seated pain. The pain is greatly increased when the patient breathes deeply with the abdomen. When the pain over the abdomen is "referred," due to diaphragmatic pleurisy, deep pressure with the flat surface of the fingers is well borne, only cutaneous hyperalgesia is present; and pinching the skin, or slightly stroking it, will elicit tenderness and pain. In chronic cases careful physical exploration of the chest will show, in most instances, signs of a thick pleura. In acute cases, the sudden onset, the severe pain in the chest and shoulder and other characteristic symptoms of pleurisy, when combined with absence of a friction sound, point to diaphragmatic localization of the lesion.

In rare cases of diaphragmatic pleurisy the diaphragm on the affected side is relaxed and strikingly elevated, and the most annoying symptom is uncontrollable hiccough.

Interlobar dry pleurisy of a tuberculous nature also occurs at times No frictions are audible, as a rule, but feeble breath sounds are found while listening over the lung above the fourth or third rib, owing to

immobilization of the lung above the affected part of the pleura. This form of pleurisy is apt to recur, as has been shown by Piéry, and after several attacks the lung is implicated in the tuberculous process. These cases of recurring interlobar pleurisy, as well as apical pleurisy which will soon be described, are characterized by symptoms of incipient phthisis without the pathognomonic physical signs and with negative sputum. Even if after several attacks the parenchyma is not implicated, the after-effects may be disagreeable, particularly when the lesion is in the left side of the chest. The thick pleura and the adhesions remain permanently and the cicatrix may contract. By their attachment to the mediastinum they may pull the heart out of its normal location outward and upward and thus hamper its action I have seen cases of this type in which no signs of excavation, or even of infiltration, of the left lung could be discovered, yet the heart was displaced, and the dyspnea, tachycardia, acrocyanosis, and debility were so pronounced as to completely disable the patient, even though there was no active parenchymatous lesion to be discerned, and the temperature had been normal for a long time. While most of these patients are below normal in weight, I have seen many who were quite corpulent, and the obesity contributed to their misery. The diagnosis is made from the history of repeated attacks of pleurisy, cardiac displacement and, very frequently, the absence of breath sounds over the upper third of the affected lung. Many of these cases are considered as non-tuberculous apical lesions. The thick interlobar pleura may at times be seen as a sharp line in the roentgenogram, and considered as an indication of a cavity. In fact many of the "annular shadows" (see p. 365) are produced by thickened interlobar plura.

Apical pleurisy is another variety of tuberculous disease limited to a portion of the pleura which is not generally appreciated to the extent it deserves. Many cases of tuberculosis with negative sputum, as well as doubtful cases in general, are in fact apical pleurisy which is not properly diagnosticated. Emil Sergent[1] and M. T. German[2] have recently made a careful study of this condition and shown that it occurs more frequently than it is diagnosticated.

It has already been shown that the pleura covering the apex of the lung is almost invariably implicated in cases of tuberculous lesions of the upper lobe of the lung. But at times the pleural lesion is, in the clinical sense, primary, and its symptoms precede those of the pulmonary lesion, or it is not at all followed by an apical process. The symptoms presented are suggestive of phthisis, but physical examination and roentgenography fail to elicit conclusive signs of a localized lung lesion, and the patient is either pronounced non-tuberculous, treated as a case of incipient phthisis with negative sputum, considered as suffering from some non-tuberculous apical lesion, etc. Many

[1] Presse médicale, 1916, **24**, 369.

[2] Étude sur le syndrome de la pleurite apicale dans le tuberculose pulmonaire, Thèse de Paris, 1916–17, No. 30.

cases of abortive tuberculosis (see p. 414) are in fact tuberculous apical pleurisy.

The onset is insidious. The patient is troubled with mild fever, unproductive cough, pain in the chest or shoulder and, coupled with anorexia, there is observed a constant loss in weight, though rapidly progressing emaciation is uncommon. I have also noted that the tachycardia characteristic of phthisis is lacking in most cases. Physical examination of the chest shows either slight or no impairment of resonance over the affected area during the early period of the illness. On auscultation the breath sounds are diminished or abolished; in some cases cog-wheel breath sounds are heard. Auscultation yields one sign which is characteristic: A friction sound is heard in the supraspinous fossa of the affected side of the chest. Its location is usually the "alarm zone," which has already been described (see p. 379). This friction sound is heard as occurring very superficially, and it is difficult to differentiate it from crepitation. It is heard only for a few days and disappears, to reappear during an exacerbation of the process. Its characteristics have been well described by Thomas Clifford Allbutt:[1] "Its significance cannot be overrated. It is far from an axiom to say that a streak of pleurisy, audible at the apex, means pulmonary tuberculosis." It is recognized by an elusive apical rub, "as if it were rather a creaking of a stiff membrane than a translation of surfaces." A faint creak may be all that is heard; it is often simulated by some fortuitous little wheeze or chirp.

The following guides may be of service in differentiating this rub from crepitation: With crepitation there is almost invariably some alteration in the breath sounds, which are either bronchovesicular or bronchial, while with a friction sound they are either feeble, or completely abolished; exceptionally there are cog-wheel breath sounds. Cough will accentuate crepitation, rarely abolish it, while a friction rub is not influenced by it. In most cases frictions are audible during both the inspiratory and expiratory phases of respiration, while crepitation is only heard during inspiration.

Sergent[2] has pointed out two other symptoms of apical pleurisy which are of great assistance in the diagnosis. They are: (1) Inequality of the pupils; (2) swelling of the glands in the supraclavicular fossa. These two signs may be found singly or in combination.

Inequality of the pupils is observed very early, and when found in a patient who coughs and shows a friction rub in the supraspinous fossa is to be taken seriously. The pupil on the side corresponding to the affected pleura is somewhat dilated. It is best observed when the patient is made to fix his gaze upon a distant dark object and it disappears when a strong light provokes a strong contraction of the iris. The extent of the pupillary dilatation varies from day to day, and in some cases it persists after the pleural lesion has healed.

[1] Lancet, 1912, **2**, 1485.

[2] Études cliniques sur la tuberculose, Paris, 1919, pp. 102, 581, 595.

The enlarged supraclavicular glands are mainly found in the angle formed by the inner extremity of the clavicle and the sternal tendon of the sternocleidomastoid muscle. If they are enlarged, light palpation of that region will reveal these glands in most cases. In patients with large muscles of the neck palpation must be done delicately while the patient has his muscles relaxed by bending his head toward the affected side. The swelling of these glands occurs late, after the disease has lasted for some time. In fact, when it does occur there are, as a rule, already signs of a parenchymatous lesion in the apex, at times even positive sputum. Occasionally the swelling is quite marked, but in most cases it is insignificant and requires careful palpation of the region before it is appreciated. Moreover, we must be careful before pronouncing palpable structures as enlarged glands. The tendon of the omohyoid, or the external jugular, may be mistaken for enlarged glands.

In a large proportion of cases patients also complain of pain in the shoulder or the back beneath the scapula. This is usually a dull pain, uninfluenced by respiration, cough, or the position of the body.

The course of apical pleurisy is mild in most cases. The patient coughs for some weeks or months and recovers. When the cough, fever, pain, etc., have disappeared the patient may feel well indefinitely, but on percussion we find that the resonance over the affected apex remains impaired and the supraclavicular fossa is more or less deeply excavated. The breath sounds remain feeble, or some sibilation may be audible. It is clear that these signs are indications that the pleura in that region has remained thick and adherent. Though no relapse has occurred, these patients are often erroneously diagnosed as tuberculous when they have common colds, or some other non-tuberculous respiratory affections, and the physician carefully examines the chest. Roentgenography may confirm the diagnosis of phthisis by showing a distinct narrowing of the pulmonary field and some opacity of the apical parenchyma; the so-called ground-glass appearance is very commonly seen, owing to thickening of the pleura and cicatricial contraction of the apex. But, as has repeatedly been stated, only constitutional symptoms should decide in these borderland cases whether the patient is sick with active phthisis requiring treatment.

Apical pleurisy is likely to recur. In some patients under my care there have been several relapses at irregular intervals, until finally the symptoms of pleurisy merged into those of active pulmonary tuberculosis—the process invaded the parenchyma and symptoms and signs of an apical lesion could be made out. It is among these cases that strictly localized lesions are encountered—the parenchyma may be completely destroyed in the upper lobe, leaving a dry cavity, and the patient recovers. The sharp line of demarcation between the diseased portion of the lung and that which has not been infected is clearly seen on the roentgenographic plate. The thickened interlobar pleura apparently limits the progress of the lesion. In others the lesion

extends, may invade the other lung, and chronic phthisis of the usual type pursues its course.

In the majority of cases, however, tuberculous apical pleurisy pursues a very benign course. The patient has slight fever for a few weeks, coughs for a variable period without expectorating, has slightly enlarged glands above the clavicle, and inequality of the pupils, while auscultation shows a friction rub over the supraspinous fossa. Within a few weeks to three months recovery may be complete, though there is likelihood of a recurrence of the trouble.

Primary Pleurisy with Effusion.—A pleural effusion is very commonly the first indication of phthisis. Numerous patients give a history of fairly good health when, after exposure, they were laid up with cough, fever, pain in the chest, dyspnea, etc. Within a few days physical exploration of the chest shows the presence of fluid in the pleura which is confirmed by an aspirating needle. It is not rare to meet with patients who say that they have felt out of sorts for some weeks, perhaps they have coughed somewhat, or have been slightly short-winded and unable to pursue their usual vocations efficiently, but still they have thought little of it. An examination reveals an effusion in one side of the thorax, though at no time have they had pain in the chest.

It is important in these cases to inquire carefully into the history of the patient. A large proportion of these "primary" pleurisies are in fact secondary to long-standing, but unrecognized, phthisis. While the patient says that he had felt quite well, interrogation often elicits the information that he had coughed for many weeks or months before the onset of the symptoms of pleurisy; perhaps that he had hemoptysis many months or years before, but had completely recovered. In fact, his physician had told him that the symptoms indicated merely a trifling derangement, a "cold," gastritis, neurasthenia, etc. But with the arrival of the new symptoms—the painful cough, the fever, the pain in the chest, the dyspnea—things took a different aspect. It is thus clear that the patient has been tuberculous for a long time, and only with the arrival of the symptoms of pleurisy with effusion he decided that he must be carefully examined. Under the circumstances, a patient with pleurisy who has been ailing for some time before the arrival of the acute symptoms is to be considered tuberculous and treated as such.

I have observed a certain number of cases of pleurisy with effusion which began with hemoptysis. In fact, in several cases the disease was ushered in with a profuse hemorrhage. All these turned out to be tuberculous. In one case the effusion was absorbed and the patient felt well for five years and then developed phthisis. It has been my practice to consider all pleurisies accompanied by hemoptysis as tuberculous.

The temperature of the patient is in most cases high, 102° to 104° F. is not uncommon. It is usually slightly remittent in type; during the

morning hours it may be one or two degrees lower than during the afternoon or evening. It is not due altogether to the absorption of toxins from the effusion, but appears to be the reaction of the body, against the invading enemy. In fact, the young, the vigorous, have higher fever than the weak, the decrepit and the aged, and in many cases the fever abates long before the absorption of the fluid. The fever is accompanied by the usual symptoms of pyrexia, anorexia, backache, insomnia, etc. After remaining high for about one or two weeks there is shown a tendency to a decline in the temperature, and within three or four weeks the patient may be completely afebrile, irrespective of the presence or absence of fluid within the pleural cavity.

The pulse is accelerated in nearly all cases, corresponding to the degree of the fever. In some cases tachycardia is severe, and a pulse-rate of 120, or more, is observed. The cyanosis, which is common to some degree in nearly all cases, may then be appalling. In rare instances in which failure of the circulation is accentuated there may be edema of the extremities; unilateral edema of the face, arm, chest, and leg, corresponding to the affected side of the pleura is occasionally observed.

Dyspnea is another symptom which is not lacking in most cases. In some it is merely objective. Though the patient believes that he is not short winded, we clearly see that he is, and the respirations are found thirty or forty per minute. During the first few days the dyspnea is often due to the pain, while later the pleural effusion, cardiac displacement, and weakness are responsible. With the beginning of absorption of the pleural fluid the dyspnea lets up and finally disappears when complete absorption has taken place.

Physical Signs.—A physical exploration of the chest shows that the affected hemothorax is larger than its mate, the intercostal spaces obliterated and, when the exudate is copious, they may even bulge out. This is in contrast with the average phthisical chest in which the intercostal spaces are deeply indented and there are inspiratory retractions to be observed. Inspection may also show *the sign of the spinal muscles* which has been described by Felix Ramond.[1] On the affected side the erector spinæ is in a state of permanent reflex contraction. On inspection the muscular mass on the affected side appears to be more prominent and broader than on the sound side. On palpation the muscles give a sensation of hardness and resistance which may be compared to that of India-rubber slightly stretched, which differs markedly from the sensation elicited in the muscles on the sound side. If disease of the spine is excluded, this is a fairly reliable sign of an effusion into the pleura. Very small effusions, which escape physical diagnosis and even roentgenography, may thus be detected.

Percussion elicits a flat note over the site of the effusion, while above the level of the fluid the note is tympanitic, owing to relaxation of the

[1] Bull. et mém. Soc. méd. d. hôp. de Paris, 1910, **29**, 747.

atelectatic lung tissue immediately above the upper level of the fluid. When the amount of fluid is small, under 500 cc, it cannot, as a rule, be detected by percussion, excepting in rare instances of very small and emaciated chests. But tidal percussion, showing immobility of one-half of the diaphragm, is suggestive when the symptoms point to pleurisy. The various lines described by Garland, Ellis, and Demoiseau may be made out by light percussion along the upper level of the fluid (Figs. 103-105). One important sign is flatness above the left hypochondrium, Traube's space, in effusion into the left pleura. In two out of three cases small effusions may be detected there early, but there are some important exceptions: It remains resonant, or tympanitic, in one out of three cases of moderate effusion. In small women with narrow chests a small effusion of about 1500 cm. of fluid may efface that space, but in large men with capacious chests a large effusion may leave it with clear resonance, especially when the patient has been kept in bed for several days and the fluid sank to the posterior aspects of the pleural cavity. I have observed many cases in which the effusion was copious, but because of old adhesions and thickening of the anterior aspect of the pleura, there was no sinus into which the fluid could penetrate anteriorly and it only filled up the chest posteriorly. On the other hand, in some cases of effusion into the right pleura, Traube's space is dull or flat on percussion owing to displacement of the left lobe of an enlarged liver downward and to the left.

The upper level of the effusion may be made out easily by light percussion. It will be found that no straight horizontal line can be drawn as in hydropneumothorax when the patient is in the upright position. As was shown by Calvin Ellis, of Boston, in 1873, "when a pleural effusion is small, it may occupy a conical section of the pleural cavity in the subaxillary region, where respiration and resonance may be wanting. But in a certain number of cases, when the effusion is quite large, if an accurate line be drawn, the flatness will be found to describe a curve gradually approaching the spine toward the base of the chest, leaving a space from one to three inches broad between the spine and the line of flatness. In this space resonance will still be detected and respiration heard." George W. Garland, experimenting on animals, confirmed the tendency of fluids in the pleural cavity to form a curved outline, the highest point of which is in the midaxillary or scapular region, declining as it proceeds forward on the anterior wall of the chest, and to a lesser degree on the posterior aspect (Figs. 103, 104 and 105). Of the various explanations which have been given for this curved line indicating the upper level of the effusion, the most plausible appears to be that while attempting to make room for itself, the fluid will compress the least resisting parts of the walls of the pleural cavity. The mediastinum, which is very mobile when not held by strong adhesions, is pushed to the opposite unaffected side. After this has reached its limits the lung will be compressed. At its roots the lung is held strongly, but at the sides the spongy tissue, when not held by adhesions, is easily com-

pressible and by retraction will recede, permitting the fluid to accumulate more along the sides of the pleural cavity. For this reason Gar-

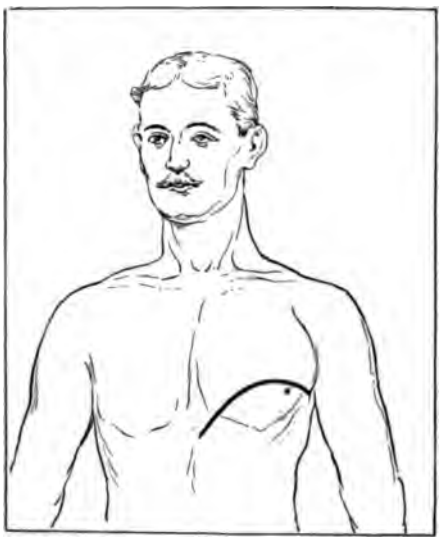


FIG. 103.—Ellis's line in pleural effusions.

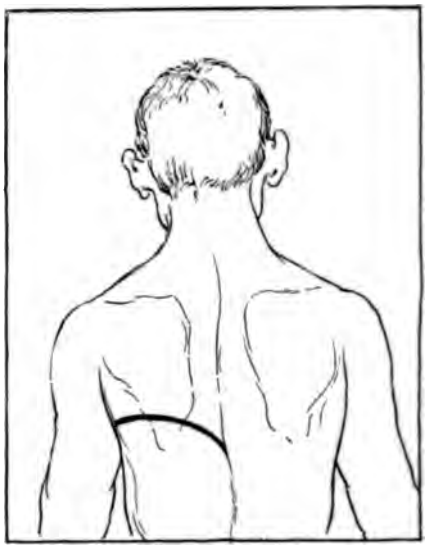


FIG. 104.—Ellis's line in pleural effusions.

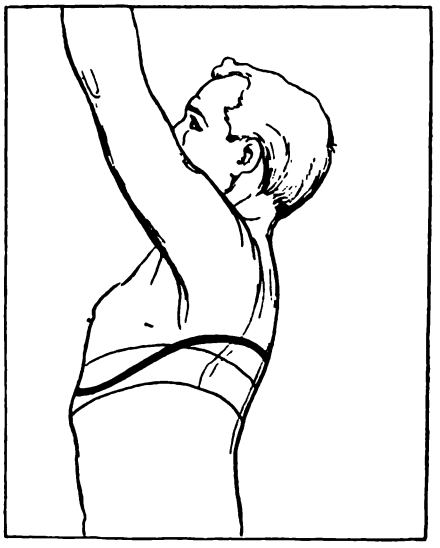


FIG. 105.—Ellis's line in pleural effusions.

land's line is only found in large effusions; when there is but little fluid, the upper level is practically horizontal. Moreover, this curve can only

be mapped out when the patient is in the erect posture; lying down produces a change in the line indicating the upper level of the fluid. In patients who walk around the line described above may be lacking—the upper level of the fluid is almost horizontal.

Many authors speak of shifting of the upper level of the fluid according to the position of the patient, and some say that it can be demonstrated in most cases. But experience has taught me that it does not occur in pleurisy, and when it is found we are dealing with hydropneumothorax. The outlook here is quite different from that in serofibrinous pleurisy. Recent investigations of this subject by H. K. Dunham[1] with the aid of roentgenography has shown that "as a general rule it can be stated that pleural effusion does not move and that movable dulness over the thorax means hydropneumothorax." There are, however, some exceptions. Soon after tapping a chest it may be observed. But here again it has been my impression that some air had entered the pleural cavity during the operation, while in many cases the lung does not completely expand soon after the fluid is withdrawn. Dunham quotes William S. Thayer to the effect that pleural effusions move when there is an old emphysema of the lung above it; and Roger Morris teaches that transudates, such as are observed in cardiac and renal diseases, will move. However, I agree with Dunham who found in more than 100 cases of pleural effusion associated with tuberculosis of the lungs, it could be demonstrated by the use of the roentgen rays that the fluid does not move as much as half an inch. This is, in fact, of immense diagnostic and prognostic importance, because if we find shifting of the dulness we are to conclude that we deal with a hydropneumothorax, or perhaps with a mediastinal neoplasm in which the prognosis is much graver than in primary tuberculous pleurisy, or in simple effusions during the course of phthisis.

Another sign of fluid in the pleura is a triangular area of dulness elicited near the spine on the unaffected side of the chest, "Grocco's triangle." In his first communication on the subject, Grocco[2] described it thus:

"Paravertebral triangle of the side opposite that of the pleural effusion. When, with a pleural effusion of sufficient size, one percusses from above downward, along the spinous processes of the vertebræ, with the patient in the sitting posture, there appears, at the level of the fluid, a dulness which, relative at first, becomes absolute as one passes downward, in association with a progressively increasing sense of resistance. In like manner, by percussing downward on the healthy side, along lines parallel to the spinous processes, there is noted, opposite the dulness in the median line, a paravertebral area of deficient resonance, of triangular shape. One side of this dull area is represented by the line of the spinous processes; another, by the lower border of

[1] Tr. Nat. Assn. Study and Prevent. of Tuberc., 1917, **13**, 181.

[2] Riv. critica di clin. med., 1902, **3**, 274; Lavori di congres. di med. int. (1902), Roma, 1903, p. 190.

the area of thoracic resonance of a short distance, which varies in length from two to three or more centimeters; the outer side is represented by a line which, starting from the base, rises obliquely to unite at an acute angle with the median line at about the upper limit of dulness. In a right-sided effusion, other things being equal, the paravertebral triangle has seemed to me more marked."

This triangle is found in nearly all cases of pleural effusion, and in rare cases of pneumonia, hydro- and pyopneumothorax, and cancer of the lung. It disappears when the patient reclines on the affected side.

Auscultation shows that the friction sound, which was audible earlier in the disease, has disappeared. The breath sounds are either feeble or completely absent in copious effusions. In cases which are followed from day to day, it may be noted that the intensity of the breath sounds diminishes, and distant bronchial or tubular breathing makes its appearance. In cases in which the effusion fills two-thirds of the affected pleura cavernous breath sounds may be heard. In patients in whom there have been signs of active pulmonary lesions, these signs may remain in the lung above the upper level of the fluid; they may become accentuated, or disappear, when the fluid is so considerable in quantity as to compress the entire lung. However, compression of the entire lung is exceedingly rare, excepting in intrathoracic neoplasms, and this is of immense importance in the differentiation of the latter from effusions due to other causes. Bronchophony is heard in large effusions, and in some cases whispered pectoriloquy, both of which are, however, also audible in other conditions involving condensation of lung tissue, or thickening of the pleura, or excavation of the lung. In some cases typical egophony may be heard, but it is at times also audible in pneumonia, and even in thickened pleura.

At times we may hear over the part of the chest filled with an effusion any kind of rales, or crepitation. They are usually derived from the catarrhal condition of the f ne and medium-sized bronchi which have not been completely compressed by the effusion. In these cases, which are not very rare, the diagnosis is often very difficult, but a consideration of the other signs, especially the displacement of the mediastinum, decides the diagnosis.

In almost all cases the vocal fremitus is absent over the site of the effusion. In very rare instances when the lung is fixed by adhesions to the posterior wall of the thorax, it may remain normal. But this is diagnostically of little value in chronic cases of tuberculosis because this is also observed in thickened pleura and intrathoracic neoplasms. The vocal fremitus may also be obliterated in cases of pulmonary infiltration when the supplying bronchus is plugged; but vigorous cough may dislodge the plug and the fremitus is again palpable.

Displacement of Organs.—In pleurisy with effusion the mediastinum is displaced toward the unaffected side of the chest, provided the exudate is ample. In effusions into the right pleura the liver may be

pushed downward and felt beneath the costal arch, while in effusions into the left side the spleen may, at times, be felt down in the abdomen, and the stomach also is often displaced downward. The weight of the fluid is sufficient to displace these abdominal organs. It is Douglass Powell's opinion that it is not the amount of fluid that is instrumental in displacing the heart in pleurisy with effusion. Rokitansky, Frank Donaldson, and others, have shown that no real pressure is exerted on the heart till the pleura is more than two-thirds filled. Small effusions may displace the heart by diminishing or abolishing the elastic retraction of the lung in the unaffected side of the chest.

Exploratory Puncture.—In nearly all cases these signs suffice to prove that there is an effusion, and its location. But this should be confirmed by exploratory puncture, both for general diagnostic purposes, as well as with a view of ascertaining the nature of the fluid. While in tuberculous pleurisy, as well as in all other conditions, exploratory puncture is a harmless procedure, yet at times we meet with some trouble, such as the conversion of a pleurisy into a hydropneumothorax. For this reason, if the general condition of the patient is good, we may leave the effusion alone. But in case the temperature continues high or hectic for more than two weeks, there are chills and, perhaps, some edema of the chest wall, especially if the patient begins to lose ground after the appearance of the exudate, an exploratory puncture should be made under strict aseptic precautions. Exploratory puncture is also indicated in doubtful cases, when differentiation between an effusion and a thick pleura is aimed at. But here when we get a dry tap we are just as much in the dark as before. In fact, in these cases of thick pleura the puncture must be made very carefully. The diaphragm is high, owing to old adhesions in various parts of the pleura, and when the needle is inserted low into the chest it may penetrate the peritoneum, the spleen, or the liver and the result is a dry tap. Large flakes of fibrin, thick pus, etc., may be the cause of a dry tap. In such cases the needle is withdrawn and reinserted in another place, but I have known of many cases in which several exploratory punctures proved negative, while incision of the chest wall showed that there was an effusion.

Exploratory puncture is also dangerous during the first few days of the appearance of the effusion. It should be avoided during the febrile stage, because it is liable to spread the tuberculous infection by producing a bacteremia. In all cases in which there is no urgency, puncture should be postponed till the fever abates.

Examination of the Exudate.—The fluid withdrawn should be placed in a sterile tube and carefully examined. The cytology of the fluid has been studied by many authors, but it appears that no positive diagnosis as to the nature of the case—tuberculous or non-tuberculous,—can be made in every case by a microscopical examination of the sediment. Some authors, notably Widal, Wolff-Eisner, and others, maintain that when mononuclear lymphocytes predominate in the sediment of the centrifuged specimen, the effusion is of tuberculous

origin, while if polynuclear leukocytes predominate, the cause is one of the pyogenic microörganisms, notably streptococci. On the other hand, D. S. Page[1] maintains that while a predominance of small mononuclear cells is usual in tuberculous effusions, they may be found in effusions accompanying intrathoracic neoplasms, or even renal disease. Page also found that coarsely granular eosinophilic cells occur in large number in non-tuberculous effusions. On the whole, I may state that a study of the cytology of pleural effusions has not been of material assistance in diagnosis.

Of more importance is an investigation of the presence or absence of mixed infection; various microörganisms should be sought, especially streptococci, staphylococci, pneumococci, etc. As will be shown later on (see p. 510), tubercle bacilli are found only exceptionally in pleural exudates. Sterile pus, which is not extremely rare in pronounced cases of phthisis with empyema, is surely of good prognostic significance. But in primary empyemata it is never found. It has been my experience that pyogenic microörganisms are found in most tuberculous pleurisies, owing undoubtedly to mixed infection. But now and then we find a case of sterile pus in tuberculous pleurisy and, as has already been stated, the prognosis is very good when this is the case, provided the pleura is not infected by repeated exploratory, or therapeutic punctures. Therefore the cytology of the fluid should be ascertained in every case. It can be accomplished very easily by centrifuging the fluid and making a smear of the sediment, staining it with Löffler's methylene blue solution.

Roentgenography.—Dry pleurisy cannot be recognized with the *x*-rays; it is only when the pleura is thickened, and perhaps calcified, that it prevents the rays from passing. Hence, acute dry pleurisy must be diagnosed clinically, while in the later stages, when adhesions form, and the membranes thicken, roentgenography is at times of great assistance in the differentiation of intrathoracic changes. On the other hand, moist pleurisy is easily recognized on the screen, or the roentgenogram by the deep homogeneous shadow it casts on the plate. Small effusions may be recognized by the fact that the diaphragm on the affected side moves less lively during respiration; in some cases it is completely immobilized. On the right side small effusions are not easily differentiated because the shadow merges with that of the liver, but in the left pleura they may be clearly shown on the screen or plate. Barjon[2] points out that immobilization precedes the arrival of the fluid and remains long after the fluid has been absorbed. It has also been shown by Weil[3] that the mobility of the diaphragm in pleurisy gives important prognostic clues. If after tapping the chest of the effusion the diaphragm is seen to resume its normal mobility, the prognosis is good; there are but slight chances that the pleural cavity

[1] Lancet, 1920, **1**, 585.
[2] Radiodiagnostic des affections pleuropulmonaires, Paris, 1916, p. 35.
[3] Presse médicale, 1920, p. 113.

PLATE XIX

FIG. 1

Localized and encapsulated purulent effusion in the right pleura. Cavity in upper lobe of the right lung. Left lung emphysematous.

FIG. 2

Interlobar effusion in the right pleura.

FIG. 3

Pneumothorax with multiple fluid levels in the left pleural cavity.

FIG. 4

Interlobar effusion in fissure between upper and middle lobe of right lung. Extensive tuberculous changes throughout right lung with cavitation.

will refill with fluid. Conversely, if the diaphragm remains immobile after the fluid has been withdrawn, fluid will in all probability reaccumulate.

In all cases of small effusions the costophrenic angle is obliterated. But it is important that the examination should be made with the patient in the erect posture, otherwise, when the amount of fluid is slight, these angles may not show any decided change.

The upper limit of fluid in the pleura is not marked by a sharp line of demarcation between the luminous lung tissue above, and the dark shadow below, as is the case in hydropneumothorax. The shadow cast by the fluid passes gradually from the deep opacity of the fluid to the transparent part above. This is due to the fact that the lung tissue immediately above the fluid is compressed and airless to a degree, and is therefore not so clear on the screen or plate as the portions higher up, where air enters freely. Garland's, Ellis's or Demoiseau's lines may be made out in many cases as a convexity of the upper level of the fluid in the axilla.

In most cases it will be noted that the brightness and clearness of the upper lobe of the lung are not so pronounced as in the opposite, unaffected, side. This should not lead to a hasty conclusion that there is a pulmonary, or perhaps a tuberculous, lesion in all such cases. Because the lung is compressed above the fluid, and often quite congested, it does not permit the rays to pass as freely as in the opposite, usually vicariously emphysematous, lung.

Another important diagnostic point needs emphasis: In nearly all, even very copious, effusions due to inflammatory causes the lung may be seen above the upper level of the fluid. When an entire hemothorax is found obscured by a shadow, showing no lung tissue anywhere, a careful inquiry should be made into the history, symptomatology, and physical signs of the case with a view of determining whether the cause of the effusion is not malignant intrathoracic disease.

The mediastinal organs, and also the trachea, are displaced in cases of pleural effusions toward the unaffected side of the chest. Even small effusions may be thus effective, provided the mediastinum is not held by adhesions. A dense shadow in the lower part of the thorax with the heart drawn toward the affected side points to a thick pleura rather than to an effusion. But to this there are frequent exceptions, which are discussed elsewhere.

Localized and encapsulated effusions are often made out with greater certainty with the *x*-rays than with physical signs. Thus, interlobar effusions are very difficult of diagnosis, but a roentgenogram shows them clearly, as a rule: A dense shadow running across the chest, while above and below it there is seen luminous lung tissue (see Fig. 4, Plate XIX). Similarly encapsulated effusions are shown clearly on the plate as dark shadows in any part of the chest (see Figs. 1 and 2, Plate XIX).

With the aid of the *x*-rays we may follow the effusion, noting care-

fully its amount, its tendencies to increase, or its absorption, and especially whether the lung shows a tendency to reëxpand after the effusion is absorbed. On these points, the roentgen rays are superior to physical diagnosis in many cases.

Course.—In non-tuberculous pleurisy the effusion is absorbed within a few weeks in the majority of cases. Only exceptionally does the fluid remain within the chest for more than two months. In tuberculous pleurisy the rule is that the effusion persists for months. Moreover, in non-tuberculous pleurisy the fever shows a tendency to abate after the effusion is tapped; often even when the fluid remains within the chest. It is different with tuberculous pleurisy. Here, tapping does not render the case afebrile; at most, it reduces it one or two degrees. In fact, in many cases the reverse is often observed. The fever may be trifling, but tapping the chest brings about an elevation of the temperature. The reason is obvious. While the effusion is within the chest, the diseased lung and with it the tuberculous lesion is compressed; toxemia is thus prevented as is the case with artificial pneumothorax. With the removal of the fluid the lung reëxpands, and moves freely with respiration, permitting reactivation of the lung lesion; hence, fever with its concomitant clinical phenomena.

It is for this reason that I am averse to tapping pleural effusions indiscriminately. I have felt that in many cases the outlook for the patient might have been better had the fluid been permitted to remain in the chest. Of course, individualization is to be practised. When the dyspnea becomes threatening, or the fever is very high, the question of tapping is to be given consideration.

In all cases in which, despite the mildness of the general symptoms, the fluid shows no tendency to absorption, tuberculosis or, more rarely, cancer of the lung, is the cause.

After the fluid has been absorbed the patient with non-tuberculous pleurisy begins to improve in general health. His appetite returns, he gains in weight and strength, and the signs in his chest may disappear at times without leaving any trace. In many cases some thickening of the pleura may be detected on physical exploration of the chest. But this is no indication that he has remained sick. It is different with tuberculous pleurisies. The effusions may persist within the chest for months. I have seen cases in which it persisted for more than two years. When it is finally absorbed, the physical signs in the chest show unmistakable signs of a tuberculous lesion in the apex of either side, while over the lower lobe, at the base, signs of thickened and adherent pleura may be easily discerned. In a large proportion of cases pleural adhesions dislocate the mediastinum toward the affected side. This is in contrast with the location of the mediastinum while the fluid was in the chest. It has been my rule to consider a thick pleura with dislocation of the heart toward the affected side as of tuberculous origin, irrespective of the constitutional symptoms presented. Still, it does not always mean *active* tuberculosis. In many cases of bron-

chiectasis, especially on the left side, dislocation of the heart is seen. These cases are usually sequels of pneumonia or pleurisy of non-tuberculous etiology. Their number has recently increased since the epidemics of influenza.

During the course of pleurisy with effusion the pulmonary apex of the unaffected side is to be watched for signs of a tuberculous lesion. Very frequently a timely diagnosis is thus made. In experimental tuberculosis of the pleura both sides are usually found affected, though the inoculation has been made only on one side, as has been shown by Robert C. Paterson. For this reason we may find an active tuberculous lesion in the lung with the unaffected pleura, though as a rule the lesion is in the lung in whose pleura the effusion is found.

Of course, the sputum is to be examined for tubercle bacilli at frequent intervals during the course of the disease.

PLEURISY DURING THE COURSE OF PHTHISIS.

As has already been stated, during the course of phthisis, the pleura is implicated sooner or later in practically every case. There is hardly a case of active, or healed, tubercle of the lung in which pathological changes cannot be made out in the pleura at the necropsy. The blood supply of the pleura, as well as its lymphatic system, shows that tubercle bacilli in the lungs, or the thoracic glands, almost inevitably must find their way into the pleura (see p. 478). Dry adhesive pleurisy is the result of the extension of the tuberculous process to the pleural membrane, in most cases. Severe cough, tugging upon these adhesions, or tearing them apart, may thus produce inflammation of the pleura. The pleurisy in such cases is, strictly speaking, of traumatic origin.

The most common variety of pleurisy in individuals suffering from chronic phthisis is the dry, adhesive form, affecting only part of the pleura, notably that overlying the affected lung area; the areas found affected in the order of their frequency being the apical, that lining the interlobar fissures, the diaphragmatic, and the mediastinal pleura. As a rule, the pleura reacts to irritants by a productive inflammation leading to adhesions of the affected areas. At times the inflammation is of the exudative variety and an effusion takes place into the pleural cavity. This effusion may be serous, serosanguineous, or purulent; it may fill the entire pleural cavity, or only part of it; it may be general, localized, or encapsulated.

Pleurisy Accompanying Acute Phthisis.—In the acute forms of pulmonary tuberculosis the pleura is usually found studded with tubercles. In most cases the effusion is rather small and negligible from the diagnostic standpoint, and in many cases it is serosanguineous. In rare instances the effusion is copious and may even mask the underlying progressive tuberculous process in the lungs, as I have seen several times. The symptoms of acute miliary tuberculosis, or of acute pneumonic phthisis, are clear cut; the patient is prostrated with high fever,

profuse sweats, rapid heart action, distressing dyspnea, cyanosis and emaciation. In the miliary cases there may be cerebral symptoms, while in the pneumonic cases distressing unproductive cough may be dominating. Severe anemia and emaciation appear early and proceed at a rapid pace. But physical exploration of the chest revealing an effusion into the pleural cavity, we are apt to be misled and consider it a simple case of pleurisy, and raise false hopes in the patient and his friends. In fact, we are justified in our favorable opinion, because it is extremely rare that a patient with a primary pleural effusion should succumb. But the fever keeps on despite tapping the pleura, and the severe constitutional symptoms do not abate. Indeed, instead of relieving the dyspnea, as is usual in many cases, tapping aggravates it.

Within a short time signs of consolidation of one of the upper lobes of the lungs will be noted; the patient begins to expectorate considerable quantities of mucopurulent sputum which, as a rule, contains tubercle bacilli. Signs of excavation soon make their appearance in either lung. In the acute miliary cases symptoms of meningitis may be the terminal phenomena.

Pleural effusions, especially serosanguineous, characterized by high fever, prostration, cyanosis, tachycardia and emaciation, should be given a guarded prognosis. If there is a history of cough, expectoration, loss in weight, etc., for some weeks or months before the onset of the acute symptoms, the cue should be taken and a careful search should be made for proofs of the underlying acute pulmonary process.

PLEURISY DURING THE COURSE OF CHRONIC PHTHISIS.

We have already stated that many of the so-called "primary" pleurisies are really secondary in the full sense of the word, because the patients had been coughing, losing in weight, sweating, etc., for some time before the appearance of the pleural symptoms. But in the vast majority of cases of pronounced chronic phthisis there are to be discerned symptoms and signs of pleurisy at one time or another. Pain in the chest during the course of tuberculosis is almost invariably due to pleurisy. As was already stated (see p. 292), the lung contains no sensory nerves and only when the pleura is implicated will the patient have pain in the chest.

Dry pleurisy of this type may be localized and circumscribed in any part of the chest, and may be bilateral. Its most common location is the apex; but the base, and especially the diaphragmatic pleura, are affected in a large proportion of cases. Usually the fibrinous exudate becomes organized, and the two sheets of the pleura are glued together by adhesions. Often large, thick strands of adhesions are seen roentgenographically, or at the necropsy, running from the diaphragmatic pleura into the de[illegible]g (Fig. 2, Plate XX). Over the apex the adhesio[illegible]g a thick fibrous shell around the dis[illegible]avations, the thick, adherent

pleura may be the only structure left instead of the upper lobe of the lung.

Symptoms of dry pleurisy may be encountered during the course of phthisis in any of its stages. The pain in the chest is felt in the neighborhood of the affected pleura, or may be referred (see p. 483) and then it is felt over the shoulder, the abdominal walls, etc. During the course of phthisis pains in the shoulder, which may become severe and intractable, should not be pronounced "rheumatic," but a careful search should be made for physical signs of diaphragmatic pleurisy. Similarly, pain in the abdomen should not be attributed to gastric ulcer, appendicitis, cholelithiasis, etc., but a search should be made for signs of diaphragmatic pleurisy. In many cases a friction sound may be heard over the affected part of the pleura, but in others the adventitious sounds emanating from the parenchymatous lesions obscure it and render it doubtful.

Symptoms.—Phthisical patients have no pains so long as the pleura is not implicated. When they get pain in the chest, they are apt to attribute it to a "cold." After a chill, or any exposure, they may feel a sharp, at times a lancinating, pain in the chest, aggravated by cough or deep breathing. The temperature, if normal before, becomes elevated to 101° F. or 102° F. Dyspnea may be distressing owing to the pain during respiratory efforts.

Inspection may be of little value in most cases, because the phthisical chest already shows lack or impairment of mobility owing to the parenchymatous tuberculous process; the same is true of percussion. Auscultation reveals a friction sound over the affected area, while the breath sounds are usually feeble. This friction sound is, at times, difficult to differentiate from adventitious sounds of intrapulmonary origin. It is, however, sufficient to bear in mind the following points: Intrapulmonary rales are usually audible as occurring during the inspiratory phase of respiration, or during the second half of inspiration, while frictions are heard during both phases, inspiration and expiration. Friction sounds are audible as if coming from a point near the bell of the stethoscope, while intrapulmonary rales appear more distant. Cough will influence the character of intrapulmonary rales, usually accentuating, rarely abolishing them, while frictions remain the same even after intense respiratory efforts by cough. At times friction sounds will become somewhat less audible after several deep inspirations, but they never thus disappear. Pressure of the bell of the stethoscope against the chest wall may intensify a friction sound, while rales remain unaffected. A friction fremitus is occasionally felt; but not as frequent as some books would indicate. A somewhat similar sensation may be felt in some cases with dry intrapulmonary sounds.

These signs are more or less easily made out when the pleural lesion is located in the lateral aspects of the chest, especially over the lower lobes of the lungs and anteriorly. But when the process affects the

pleura over the apex of the lung, over the diaphragm, of the mediastinum, it may be difficult to localize the pleural lesion. Over the apex, friction sounds may be easily mistaken for small, moist rales, or crepitation, and some authors have been inclined to attribute most of the above-mentioned sounds, when heard over the apex, to friction sounds, alleging that incipient phthisis is always accompanied by pleurisy, and the sounds are due to frictions (see p. 346). Deep breathing, however, will accentuate intrapulmonary sounds, while frictions are not thus influenced. Feeble breath sounds speak in favor of pleurisy, especially when the pleura is thick. Crepitation is almost invariably accompanied by bronchovesicular or bronchial breathing. With feeble breath sounds large, moist, consonating rales are invariably of intrapulmonary origin.

Pleural Adhesions.—Dry pleurisy in pronounced phthisical subjects has an important influence on the course of the underlying disease of the pulmonary parenchyma. In addition to the painful suffering it inflicts it is liable to terminate in an effusion. But this is exceptional. In most cases adhesions result. Very often, by limiting the motion of the affected parenchyma, as well as through some as yet not understood biochemical and immunological processes, these exudates and adhesions impede the progress of the tuberculous lesion in the lung, retard the progress of the disease, and improve the prognosis in general.

In most cases the adhesions are limited to the area of the pleura immediately overlying the diseased part of the parenchyma of the lung. The diagnosis may be made by paying attention to the following points: On inspection the affected area of the chest is seen to move but slightly during respiration; the motion may be restricted, or there may be lagging over a limited part of the chest wall. Instead of expanding during inspiration, the intercostal spaces will be seen to retract during each filling of the chest. These inspiratory retractions are of immense diagnostic importance, but they are not infallible. They may be seen in cases without adhesions—when there is airless lung tissue with a thick visceral pleura, and this is not rare in chronic phthisis. Very frequently enlarged venules may be seen on the chest wall, indicating interference with the circulation by compression of the venous flow at the affected area. Moreover, owing to the retraction of the upper part of the pleura and lung in apical adhesions, the supra- and infraclavicular fossæ are deeply excavated. When the basal pleura is adherent, the lower part of the chest appears smaller and expands to a lesser degree than the opposite side. Percussion elicits an impaired note, frequently with a tympanitic overtone, especially when the apical pleura is affected. Over the base the note may be flat and, because the vocal fremitus is absent or defective, fluid is thought of. Friction rales are at times heard over the apex; feeble breath sounds are the rule. Loud, consonating rales and clicks of intrapulmonary origin may be so pronounced as to overshadow all other sounds. It is these adhesions over the apical pleura that interfere with the success of therapeutic

pneumothorax in many cases. They form a thick, unyielding shell around the diseased apex of the lung, and do not permit it to collapse, or to be compressed by the air which enters the pleural cavity covering the lower, unaffected lobes.

When the pleura over the lower lobe is affected by adhesions, percussion may yield a normal, or even slightly tympanitic note when it is not much thickened. The breath sounds are almost invariably feeble in the lung under an adherent pleura, and tidal percussion shows that the affected side does not expand as efficiently as the opposite side, and that the diaphragm also does not move properly. Inspiratory retractions, while not pathognomonic, yet they are so common in pleural adhesions that they should be looked for in every suspicious case. However, our experience with the production of therapeutic pneumothorax shows clearly that *there are no absolutely reliable signs of pleural adhesions. Even roentgenography fails very frequently.* In cases in which all the signs point to adhesions, a pneumothorax may be induced at times with ease; while in others, in which all the signs point in the direction of a pleura free from adhesions, all attempts at introducing gas fail (see Chapter XLIV). It seems to me that only pleural adhesions with thick pleura, especially a thick parietal pleura, may be diagnosed, but there may be strong adhesions without perceptibly thickening the pleura, and it is in these cases that we fail frequently. It also depends on which pleura is thick. If it is the visceral pleura—most frequently the one affected—we may find signs pointing to adhesions which, in fact, do not exist. When the parietal pleura is thickened, we almost invariably will find the adhesions by the usual methods.

Roentgenography in the Diagnosis of Pleurisy and Adhesions.—Small circumscribed adhesions of the pleura are not recognized with the *x*-rays. In most cases with thick, adherent pleura over the tuberculous apex it is impossible to state with any degree of positiveness whether the pleura is thick and adherent or not, because of the abnormal shadows produced by the parenchymatous lesion. When extensive and massive, a thick pleura may be recognizable, especially when the membrane over the lower lobes is affected. We then note that the convexity of diaphragm is no more a smooth line sharply demarcating it from the luminous lung tissue, but that it is uneven and deformed, and various bands of connective tissue may be noted projecting into the pulmonary parenchyma. The costodiaphragmatic sinus and the cardiohepatic angle are either obtuse or completely obliterated (Fig. 1, Plate XVI). The motion of the diaphragm is restricted or abolished.

In older cases, with more extensive adhesions, the condition may be recognized at first glance on the screen or plate. The ribs in the affected side form a very acute angle descending from the spine, the intercostal spaces are narrower than those on the opposite side, the luminous lung area is of smaller extent, owing to pulmonary retraction, than on the opposite, unaffected side. The mediastinum is pulled to the affected side. The diaphragm is immobile and often elevated.

Because of compensatory emphysema, the luminosity of the lung in the unaffected side is more pronounced than would be expected.

In many cases of extensive pleural thickening and adhesions of the lower part of the chest, it is difficult to differentiate this condition from fluid in the pleural cavity, as has been intimated above. Usually the *x*-rays clear up the diagnosis. In fluid the intercostal spaces are wider, the mediastinum pushed to the unaffected side, etc. But we frequently meet with cases in which it is very difficult, or impossible, to decide as to what we are dealing with, with all diagnostic means at our command. Even exploratory puncture, when it turns out negative, may not clear up the diagnosis. At times it is difficult to decide whether dulness and the shadow on the plate found in the lower part of the chest are due to a thick pleura or to a parenchymatous lesion. As a rule, when the percussion note is dull or flat, and the *x*-rays do not show a deep shadow, the lesion is probably pleural; conversely, when the percussion note is but slightly impaired, or has a tympanitic overtone, we are, in all probability, dealing with a parenchymatous lesion. But even to this there are many exceptions.

A thickened interlobar pleura cannot be diagnosed except with the aid of roentgenoscopy. But it may also be missed in the roentgenogram, unless the tube is placed high, on a level with the patient's head so that the rays pass through the chest at an oblique angle, from above downward through the whole width of the thickened interlobar septum, thus casting a shadow of its widest and thickest surface. This is best accomplished by placing the tube on a level with the patient's head when viewing the chest anteriorly, and on a level with the sacrum when viewing the patient at his posterior aspect. Its appearance can be seen on Fig. 1, Plate XX.

At times we meet with interlobar effusions which may be easily recognized by their physical signs—a transverse band of dulness running across the chest along the second and third interspace, while above and below the resonance is clear. This, in addition to bronchial and, more commonly, feeble or absent breath sounds, and whispered pectoriloquy, should excite suspicion of an interlobar effusion when there are also symptoms of pleurisy, such as pain in the chest, fever, cough, etc. But after all the diagnosis is made positively only with the aid of the *x*-rays. But even here an intrathoracic neoplasm is at times mistaken for an interlobar effusion, and *vice versa*.

On the screen or the plate (Fig. 4, Plate XIX) there will be seen an opaque band running across the chest below the second and above the fourth or fifth ribs. The lung is divided into three regions: The upper is more or less bright, the middle, dark, and the lower again bright. In the fluoroscope this suspended shadow may be seen moving with the respiratory movements of the chest, while a neoplasm is, as a rule, immobile. The motion of the diaphragm is practically normal. An intrathoracic neoplasm may also produce such a picture on the screen, but it is differentiated from an interlobar effusion by the clinical history of the case.

Pleural Effusions during the Course of Phthisis.—In most cases the implication of the pleura in the tuberculous process passes away leaving adhesions and, at times, without leaving any obvious traces behind. Some patients thus suffer from recurrent attacks of dry pleurisy, so long as the tuberculous process in the lung remains active. In others, effusions occur. This may be observed during any stage of the disease. When occurring before the recognition of the lung lesion, we are apt to consider it as "primary" pleurisy, but careful inquiry into the past history of the patient shows the fallacy of such an assumption (see p. 489). The effusion may be serous, serofibrinous, serosanguineous, or purulent. When it is serous the fluid can hardly be distinguished from that found in non-tuberculous cases. As will be shown later on, tubercle bacilli can only rarely be demonstrated in the exudate, and implantation on cultures, as well as inoculation experiments, are too often negative to be of real diagnostic value. In the serosanguineous exudates the chances of finding tubercle bacilli are greater than in purely serous, or serofibrinous fluid.

Hemorrhagic effusions occur mainly in tuberculosis, but may also be encountered in cancer of the lungs or pleura, in traumatic pleurisy and in pleurisy affecting persons suffering from certain cachectic conditions, notably scurvy, in certain exanthematous diseases, as hemorrhagic smallpox, and, exceptionally, in persons suffering from cirrhosis of the liver, aneurism of the aorta, and even chronic nephritis.

In tuberculosis of the pleura the blood is derived from the rich network of bloodvessels which are at times seen in these processes, especially where there is a false membrane. The physical signs and the symptoms of serosanguineous pleurisy are not different from those found in cases with serous effusions. It is only by exploratory puncture that the diagnosis is made. But we must guard against certain sources of error. While performing exploratory puncture with a thick needle a bloodvessel may be injured and bloody fluid is seen in the barrel of the syringe, though within the pleura it is clearly serous. In some of these cases it may be noted that the first part of the fluid entering the syringe is bloody, then it becomes paler, and the final part is practically straw-colored. Rarely the reverse is observed: The first portion is serous, while at the end it becomes sanguineous, evidently because the needle touched a bloodvessel. Moreover, after one exploratory puncture, especially after tapping the chest, when serous fluid is removed, a second puncture, performed some time later, may show the fluid sanguineous even when there is no malignant disease nor tubercle of the pleura. The blood is then distinctly of traumatic origin. These cases are responsible for the numerous instances one encounters in which sanguineous fluid was found in the chest and no symptoms of tuberculosis or cancer are subsequently observed to follow. In my experience sanguineous fluid is mainly found in very acute cases of pulmonary tuberculosis, and only exceptionally in chronic cases.

We have thus, in most cases of bloody fluid, to differentiate between

cancer and tubercle. When due to malignancy, the history will show a slow onset, with little or no rise in the temperature. In some cases there may be found a relatively large number of coarsely granular eosinophile cells or corpuscles in the aspirated bloody fluid. In tuberculous pleurisy with effusion the history points to an old tuberculous process, and there is marked pyrexia, excepting in the rare cases of latent effusions. Microscopical examination of the fluid shows a high lymphocyte count, in addition to the abundance of red blood corpuscles. But, as was already stated, the cytology of the fluid is not reliable diagnostically.

Hemorrhagic effusion occurring during the course of phthisis may remain within the pleura for a long time. I have seen cases in which it remained for longer than two years. In rare instances tapping once or twice will free the pleural cavity of the fluid, but in the vast majority the exudate reaccumulates. In rare cases the pressure effects —dyspnea, cyanosis, edema of the extremities, etc.—are instrumental in bringing about a fatal issue; in others, the tuberculous lesion in the lung, or some other complication sooner or later relieves the patient of his earthly sufferings.

Purulent effusions are comparatively infrequent during the course of phthisis. Whenever they occur I am suspicious that a latent pneumothorax has existed; and pneumothorax is frequently overlooked. I have recently paid special attention to this point, and *in the majority of cases of empyema in phthisical subjects I have been able to discover roentgenographic evidence of an air pouch above the level of the fluid.* Empyemata are thus due to rupture of the visceral pleura at some point, be the loss of continuity ever so minute, and the entry of air, as well as secretions from the diseased lung into the pleural cavity. On the other hand, it is possible that empyema may occur in phthisical individuals without rupture of the pleura; the cases in which the pus is practically sterile testify to this. Similarly, during epidemics of acute respiratory infections tuberculous patients are often affected and empyema at times follows. I have seen several cases of encapsulated empyema in phthisical subjects following intercurrent influenza; one following an operation on the tonsils. The etiological agent in these cases is usually one of the various strains of pneumococci or streptococci.

Symptoms.—The onset of pleurisy with effusions may be abrupt, as in primary cases. The patient has been getting along with his tuberculosis quite well, or has been improving, when he is seized with pain in the chest, dyspnea, and cough. In other cases pains in the chest have been repeatedly felt by the patient, and recurrent dry pleurisy has been diagnosticated. But now there is noted an increase in the dyspnea, while coincidentally the pain in the chest disappeared. We also meet with patients who give no history of any extraordinary symptoms, but an examination of the chest reveals an effusion. These *latent pleurisies* are not very rare in phthisical subjects. Fever is the

rule, but during the course of active phthisis this cannot guide us because of its almost invariable presence in these patients. In afebrile tuberculous patients there is noted an elevation in the temperature with the arrival of fluid in the chest.

The effusion in the pleural cavity is easily recognized by the physical signs, and exploratory puncture which have already been detailed above (see p. 490). But in phthisical patients localized effusions are very frequent, because old adhesions limit the size of the exudate. In addition to the interlobar exudates which have already been mentioned (see p. 497) there may be localized effusions in any part of the pleural cavity, most commonly in the pleura lining the lower lobes. (See Fig. 1, Plate XIX.) In these cases exploratory punctures are to be made with circumspection. The site of the exudate should be clearly delimited before the needle is inserted, and the *x*-rays should be used freely.

Serous and serofibrinous exudates are apt to remain a long time in the chest of tuberculous patients, though we often meet with cases in which the fluid is absorbed within a few weeks. Purulent exudates, on the other hand, remain indefinitely, though I have seen several cases in which the pus broke through a bronchus and was expectorated, the patient improving. But it is to be emphasized that this is merely a possibility. In most cases the fever keeps at a high level, is often hectic, characterized by frequent chills, severe emaciation, amyloid degeneration of the liver, spleen, kidneys, intestines, etc., and the patient finally succumbs to exhaustion.

The onset of purulent effusions in tuberculous subjects may also be very insidious. The patient has felt quite well, but of late has begun to lose ground; has had chills, hectic fever, nightsweats, dyspnea, etc. In some patients under my observation the fever was slight, there were no pains in the chest, and the cough was mild. But they had been losing in weight and strength. An examination of the chest reveals the presence of an effusion which, because of the mildness of the general symptom, is thought to be serous. But an exploratory puncture shows the presence of pus in the pleura. Considering the difference in the prognosis and treatment of serous as compared with purulent effusions, it is clear that in every case the nature of the fluid should be ascertained by exploratory puncture—the only way in which we may inform ourselves as to the character of a pleural effusion. In the majority of cases of empyema careful inquiry elicits a history strongly suggestive of a latent pneumothorax. In these cases the condition is, in fact, that of pyopneumothorax.

PROGNOSIS IN TUBERCULOUS PLEURISY.

As is well known, *the immediate outlook in tuberculous pleurisy is very bright in nearly all cases.* It is the ultimate outlook which is of importance. The problems are: Will the patient, recovering from an

attack of pleurisy, sooner or later develop active pulmonary tuberculosis? If he does, will the tuberculous process be of a progressive and dangerous type, or will it run a slow benign course? The seriousness of these prognostic problems is realized by every physician whenever he has a case of pleurisy under his care. The entire future of his patient depends on this ultimate prognosis of pleurisy.

Prognosis in Primary Pleurisy.—*In dry pleurisy the immediate outlook is almost invariably good.* Within a few days, at most two weeks, the fever, cough, dyspnea, etc., abate, the pain diminishes in intensity and finally disappears, and the patient may be considered well. In many cases the friction sound is audible in the chest for a long time; I have found it in patients many months after an acute attack, but usually it disappears within several weeks. The pains in the chest at times remain indefinitely; they are apt to appear during sudden changes in the weather, but are usually not severe enough to disable the patient.

In rare cases of dry pleurisy in young persons strong and dense adhesions of the pleural sheets are formed, and deformities of the chest may result, localized retractions of the chest wall may be noted, displacement of the mediastinal organs may occur, and dyspnea may torture the patient, especially in left-sided interlobar pleurisy. In others, with basal dry pleurisy, the diaphragm remains elevated and more or less immobilized, and some local bronchiectasis remains permanently. The result is that the patient keeps on coughing and expectorating for many years, perhaps for life. The prognosis is that of bronchiectasis, but the patient is likely to be told by some physicians that he is tuberculous with a basal lesion. Many of these patients are sent to sanatoriums during each exacerbation of the cough, expectoration, etc. In others, an acute attack simulates pneumonia. Many state that they had had several attacks of "pneumonia," which, in fact, were acute exacerbations of the bronchiectasis.

Dry pleurisy is likely to recur. One who has had one attack, as a rule, suffers from repeated attacks at irregular intervals. In these cases the pleura remains thickened, the mediastinum displaced, and bronchiectasis develops in any part of the lung, most commonly the base. In others, the patient recovers from the first few attacks, but finally develops pulmonary tuberculosis. *Recurrent attacks of dry pleurisy are therefore to be considered as a sure sign of tuberculosis, and treated as such.* Especially is this true of apical pleurisy.

Prognosis in Pleurisy with Effusion.—A pleural effusion is usually preceded by an attack of "dry" pleurisy. In fact, in all cases of dry pleurisy our immediate prognosis is to be guarded; we should wait some days, watchful for the appearance of fluid. When an exudate in the pleura is made out, the prognosis is not markedly aggravated. *Death of a patient with a pleural effusion is extremely rare,* especially now when, by tapping, we can avoid accidents and menacing symptoms due to overfilling of the pleura by the fluid.

Usually small or moderate-sized effusions are absorbed within a few weeks and the patient recovers. In many cases, fully four-fifths, the disappearance of the fluid leaves the patient in excellent condition; he soon regains his lost weight and strength, and an examination of his chest several months later may not show any traces of the disease which he passed through. In a large proportion of cases pleural thickening and adhesions remain. In some, bronchiectatic conditions remain indefinitely, manifesting themselves by periodical attacks of fever, cough, and expectoration, which may be influenced by the posture of the patient. In still others, the adhesions and sclerosis are instrumental in producing displacement of the heart, and dyspnea is a permanent feature which keeps them troubled for life.

But the fluid may not be absorbed so soon. It may remain in the chest for many months. I have seen cases in which the fluid was not absorbed for over two years; to remain within the pleural cavity for two or three months is a common observation. In these cases tapping is of little or no avail; within a few days the fluid reaccumulates and the symptoms of intrathoracic pressure reappear. As a rule, when the fluid has been in the chest for a long time the fever abates; rarely we find a chest full of fluid in a patient with a normal temperature. He only coughs, and is more or less short-winded. In some of these the tapping may result in a return of the fever, cough, etc., to be ameliorated, or disappear when the exudate reaccumulates.

Of course, when a serofibrinous exudate becomes purulent from any cause, which happens but rarely, the prognosis is much aggravated. If the patient has an active tuberculous lesion in the lung, the prognosis is very grave indeed. Recovery is exceptional.

I have seen one case in which a serofibrinous effusion broke through a bronchus, overfilled the lung, and nearly suffocated the patient. It is noteworthy that no infection of the pleura took place. The patient recovered after expectorating the fluid from his chest.

At times we observe that tuberculous pleurisy with effusion spreads to other serous membranes—the pericardium, the peritoneum, and finally to the meninges. Some authors hold that many cases of *polyserositis* are of tuberculous origin. A comparatively large proportion of patients show, as one of the terminal phenomena, symptoms and signs of tuberculous peritonitis with ascites; meningitis also occurs in rare instances. In a recent study of this subject, P. Ameuille[1] has suggested that in such cases the infective agent may be a strain of tubercle bacilli which has special affinity for serous membranes. But this requires further proof.

There remains yet to be mentioned that in very rare instances sudden death terminates a case of pleural effusion. The patient, without any premonitory symptoms, perceives agonizing pain in the pectoral region, severe dyspnea, becomes cyanosed, and dies. The causes which have

[1] Am. de méd., 1917, **4**, 55.

been considered as operative in these cases are: Kinking of the vena cava in left-sided effusions; pressure on the right auricle in right-sided effusions; embolism of the pulmonary veins, or the brain, the result of thrombosis in the pulmonary vessels. But in some cases none of these, and other suggested factors, explained the sudden death. In extremely rare instances tapping, or even simple exploratory puncture, is followed by sudden death.

Are All Pleurisies Tuberculous?—The most important prognostic problem in these cases is whether the patient, after recovering from his pleurisy, will develop pulmonary tuberculosis, and if so, what effect will the pleural lesion have on the immediate and ultimate outlook for recovery. Experience has taught that a large proportion of patients with pleurisy ultimately develop phthisis. But we also know many who have remained alive and well for many years, or for natural life. For these reasons physicians warn their patients with pleurisy that it is not enough to treat the primary disease, but that it is absolutely imperative to take into consideration their chances of becoming phthisical. I know of many persons suffering because this possibility has been imparted to them by their physicians; they feel as if the sword of Damocles is hanging over their heads.

It is therefore important to be able to single out the patients who are likely to become tuberculous ultimately, and those who are not. This we are not able to do in every instance, but there are indications which clearly show us the way in a large proportion of cases. The tuberculous nature of pleurisy may be determined by the following considerations:

1. Tubercle bacilli may be found in the exudate removed with an aspirating syringe, or by tapping.

2. The symptoms presented by the patient during the pleural disease, as well as soon after recovery.

Tubercle Bacilli in the Pleural Exudate.—Tubercle bacilli are only rarely demonstrated microscopically in pleural exudates. Even in cases of active and progressive phthisis, the fluid is at times sterile, and in many cases in which microörganisms are found, they are usually the germs of pneumococci, staphylococci, streptococci, etc. Thus, Ehrlich found tubercle bacilli in pleural exudates only in 2 out of 22 cases; Longa and Pensunti, in 1 out of 22; Jakowski in 1 out of 32; Fernet, in 3 out of 20; Thue, in 1 out of 30; Weber, in 1 out of 23; Landouzy and Queryat found them only once in their extensive experience. Netter,[1] collating these figures, shows that in a total of 415 cases of serofibrinous pleurisy he found an average of 2 per cent in which tubercle bacilli could be demonstrated in the exudate microscopically. In my own cases it is extremely rare to find them.

But it appears that the negative outcome of microscopic examination of the exudate does not exclude the possibility of tuberculosis as a cause of the pleurisy. The fact that in cases with pronounced and

[1] Thèse de Paris, 1883; Bull. soc. de méd. des hôp., 1891, p. 176.

advanced tuberculous lesions in the lungs no bacilli are found in the fluid, shows that there are some factors which either destroy the bacilli within the fluid, or interfere with their staining proclivities. Even though it has been my impression that many pleural effusions complicating pulmonary tuberculosis are caused by pyogenic microörganisms, or pneumococci, as is attested during epidemics occurring in hospitals for consumptives, as for instance influenza, yet a larger proportion than 2 per cent is undoubtedly due primarily to tubercle bacilli.

Attempts at culturing the fluid on proper media have also failed to show the presence of tubercle bacilli in the majority of specimens examined. Similar unsatisfactory results have been obtained by inoculation experiments; only 10 to 20 per cent of the pleural exudates inoculated into animals have proved positive, as can be seen from the extensive statistics gathered by Chantemesse and Courcoux.[1] With improved methods, the proportion of positive results has not been materially increased. It has namely been found that when a large quantity of the aspirated exudate is injected into a guinea-pig, it is more likely that the animal should become tuberculous than when a small quantity is injected. But even with the injection of 30 cc of the fluid, or its centrifuged sediment, the results more often turn out negative than positive.

Only recently we have been gleaning some light on this intensely interesting, and also very practical, problem. In experimental tuberculous pleurisy with effusion, tubercle bacilli are only rarely discovered. Robert C. Paterson[2] found that about two hours after inoculating the pleura of a guinea-pig very few, or no bacilli, either phagocyted or free, could be discovered in the effusion. He found, however, that these same effusions were virulent for, and actually infected, normal guinea-pigs in every case when inoculated subcutaneously. The problem then arises: What becomes of the baccilli in the effusion? It seems that in other serous membranes the bacilli also disappear. Thus, it is very rare that tubercle bacilli are found in the ascitic fluid in peritoneal tuberculosis, and in cerebrospinal fluid in tuberculous meningitis. Rist, Roland, and Kindberg[3] found most of their peritoneal inoculation experiments turned out negative, while Manwaring and Bronfenbrenner[4] observed that the bacilli disappeared from the peritoneal exudates in sensitized animals. The exudate of serous membranes is thus apparently bactericidal.

It has also been suggested that the bacilli are too few in number to be found with ease in the fluid microscopically; that those which are present are enmeshed in flakes of fibrin. For this reason, large quantities of the fluid, or better of the centrifuged sediment, may produce infection after inoculation, while small quantities fail.

[1] Les pleuresies tuberculeuses, Paris, 1913, p. 12.
[2] Am. Rev. Tuberc., 1917, **1**, 353.
[3] Ann. de méd., 1914, **1**, 312, 375.
[4] Jour. Exper. Med., 1913, **18**, 601.

But after all, it seems that serous surfaces, excepting that of the meninges, react very favorably to infections, particularly with tubercle bacilli. Thus, tuberculous joints show strong tendencies to heal; so does the peritoneum. The same is true of the pleura. Most tuberculous infections of that serous membrane lead but to dry pleurisy, or to small, insignificant effusions which are spontaneously and often quickly absorbed. Even in cases in which the entire pleura is involved in the process, the prognosis is good in nearly all cases, as was already shown (see p. 508). For this reason some authors have been inclined to attribute all the so-called primary pleurisies to an attenuated strain of tubercle bacilli. It is, however, the opinion of other writers, notably Königer, that the attenuation in the virulence is due to the action of the exudate produced by the reacting pleura. Whether this is due especially to active antigens or antibodies, or to the very strong capacity of the pleura to absorb foreign material, cannot be stated with any degree of exactitude at present. This is a point which deserves further careful investigation.

On the whole, it may be stated that irrespective of the cause, *absence of tubercle bacilli from pleural exudates, as indicated by microscopic examination or inoculation experiments, by no means shows that the lesion in the pleura is of a non-tuberculous character.* This is of immense clinical importance for obvious reasons.

Clinical Facts about the Tuberculous Origin of Pleurisy.—For more than a century physicians have suspected that most of the inflammatory processes in the pleura, when not due to another obvious cause, such as an intrathoracic neoplasm, or to cardiac or renal disease, are of tuberculous origin. Stoll, in the latter half of the eighteenth century, already considered latent pleurisy as tuberculous. Bayle said that "pleurisy is really not a cause, but an effect of tuberculosis." Laennec was very emphatic when referring to the tuberculous nature of pleurisy. "It is absurd," he said, "to believe that tuberculosis may terminate in pleurisy; the facts of pathological anatomy show that in the vast majority of cases tuberculosis may be latent for a certain time and cause no deviation from normal health, while in other cases pleurisy is but the first manifestation, often really the effect, of the presence of tubercle which existed within the body for some time."

Modern clinicians are inclined to the same view. In this country the first to collect a series of cases which were under observation for a long period of time was Vincent Y. Bowditch[1] of Boston, who found that out of 90 cases of acute pleurisy which had been observed by his father and followed up by himself between 1849 and 1879, 32 died of, or had, phthisis. George G. Sears[2] collected the following figures from the literature: Of 451 cases of pleurisy, 176, or about 39 per cent, subsequently developed phthisis or other well-marked tuberculous

[1] Tr. Am. Climatol. Assn., 1889, **6**, 1.
[2] Boston Med. and Surg. Jour., 1892, **126**, 192.

affections. Barr[1] found that out of 57 cases of pleurisy between 1880 and 1884, 21 had already died of some form of tuberculosis, mainly pulmonary phthisis, at the time his report was made (1890). Couston and Dubrull,[2] from army experience, say that all soldiers who have suffered from pleurisy are no longer fit for military duty, and that a majority die later from tuberculosis. William Osler[3] reports that among 86 cases in his wards in which the after-histories were studied by Dr. Hamman, 34.8 per cent became tuberculous and died. In his Shattuck lecture[4] he reports that he had carefully analyzed the post-mortem records of his ward cases in which pleurisy—fibrinous, sero-fibrinous, hemorrhagic, or purulent—was found and the result was that 32 were definitely tuberculous. The after-histories of 130 cases of primary pleurisy with effusion reported by Hedges[5] showed that at least 40 per cent died from or had tuberculosis within six years.

The most extensive series of cases carefully analyzed were reported by Allard and Köster.[6] Allard deals with 200 cases of idiopathic pleurisy treated from 1881 to 1893, their subsequent fate having been investigated in 1900. Köster deals with 371 cases of idiopathic pleurisy, and 62 of specific pleurisy, treated from 1894 to 1908, and reported in 1910. They also made an analysis of 2123 cases of pulmonary tuberculosis as to the frequency of pleurisy in their past history. The two series were compiled along the same lines, but independently of each other. In the first series, representing 180 cases of serous and 20 of dry pleurisy, it was found that sixteen to twenty-eight years later 87 patients were alive and well; 28 were tuberculous, 61 had died of tuberculosis and 24 had died from other causes. In the second series, representing 334 cases of serous, and 37 of dry pleurisy, it was found that two to sixteen years later 164 were alive and well, 118 were tuberculous, 62 had died of tuberculosis, and 27 had died from other causes. Taking the two series together, the writers find that idiopathic serous pleurisy is followed sooner or later by pulmonary tuberculosis in 47.7 per cent of cases, and that even in cases of idiopathic dry pleurisy the percentage is as high as 42.

It has also been found that a rather high proportion of tuberculous patients have had pleurisy before the onset of their pulmonary disease. Thus Allard and Köster report that among 2123 cases of phthisis 650, or 30.6 per cent, gave a history of idiopathic pleurisy. E. A. Pierce[7] analyzed two series of cases dating from 1905 until the time of his report. In the first series of 1767 cases of pulmonary tuberculosis, 614, or 35 per cent, gave a history of pleurisy. In the second series of 518 cases, 52 per cent gave a history of previous pleurisy. He adds that,

[1] British Med. Jour., 1890, **2**, 1058.
[2] Gaz. hebd. de méd., 1886, **23**, 662.
[3] British Med. Jour., 1904, **2**, 999.
[4] Tr. Massachusetts Med. Soc., 1893.
[5] St. Bartholomew's Hosp. Rep., 1900, **36**, 83.
[6] Hygeia, 1911, **73**, 1105.
[7] Northwest Med., 1918, **16**, 79.

including simple adhesions with other marked changes, pleurisy was found in 74.4 per cent of 215 cases.

Statistics like the above, indicating that from 30 to 40 per cent of patients suffering from pleurisy subsequently develop phthisis, or that from one-third to more than one-half the tuberculous patients have had pleurisy before the onset of the pulmonary tuberculosis, abound in medical literature. But it appears that not all clinicians have had the same experience; many report that, while pleurisy is often followed by phthisis, the proportion is not so high as the above statistics might lead us to suppose. Thus, Blakiston[1] reports 53 cases which had remained well for several years; Austin Flint speaks of 47 cases with but 3 possible instances of subsequent tuberculosis. Out of 21 cases reported by J. P. Bramwell[2] only 3 died of tuberculous disease. Coriveaud[3] had but 4 deaths from this cause out of 27 cases, 1 of whom he had followed for twenty-five years and 1 for fifteen.

That the menace of pleurisy, however significant, is not threatening every patient, is attested by the experience of physicians of long years in practice; they all have many patients who have had pleurisy, dry and with effusion, and remained well for years. To be sure, in hospital practice we encounter patients who have become tuberculous after pleurisy, but those who remain well do not come into hospitals. It is therefore important to bear in mind that while a large proportion of cases of pleurisy is due to tubercle, not all cases are, and not everyone develops active and progressive tuberculosis subsequent to an attack of pleurisy. In fact, *more than three-fifths the number of patients with pleurisy pass through life without developing phthisis,* as the statistics cited indicate. The reasons for this are to be sought for in the following facts: (1) Many cases of pleurisy are due to microörganisms other than the tubercle bacillus, or altogether due to non-specific causes; (2) even when due to the tubercle bacillus, active pulmonary tuberculosis does not follow in all cases, and when it does, the outlook is not so gloomy as some statistics seem to show.

Non-specific Pleurisy.—Pleurisy may be produced experimentally by the injection of irritants into the pleural cavity, especially turpentine. Injuries to the chest also are often instrumental in producing pleurisy. Fractured ribs, and the calluses which are produced while they heal, are, at times, responsible for pleurisy which is clinically recurring, producing symptoms at irregular intervals, not unlike those of dry pleurisy due to other causes. These may be considered aseptic pleurisies; though the ends of the fractured ribs, or the callus, may act as irritants and reduce the vitality of the pleural tissue, thus favoring the localization of bacteria brought there hematogenously. Still they cannot be considered specific. Similarly, pleurisy is very common in cases of cancer of the thoracic viscera, and in certain cases of cardiac

[1] Quoted from Sears.
[2] Edinburgh Med. Jour., 1889, **2**, 909.
[3] Jour. de méd. de Bordeaux, 1887–8, **17**, 601.

and renal disease. Though they are not of an inflammatory character, yet they produce effusions.

Among the pathogenic microörganisms, the tubercle bacillus is not alone holding the evil distinction of producing pleurisy. Thus, the metapneumonic pleurisies are very common, and those due to various strains of streptococci and staphylococci, influenza bacilli, etc., which at times occur in epidemics, cannot be considered tuberculous. I have been under the impression that the last-mentioned pathogenic agents are quite frequently responsible for pleurisy in tuberculous patients, occurring as it does occasionally almost epidemically in hospitals for consumptives. On the other hand, considering the wide distribution of tuberculosis in mankind, it is to be expected that many with dormant or latent tuberculous lesions should have them reactivated during or after attacks of pleurisy due to any cause. When judging statistics of this sort, this factor is to be borne in mind.

Factors Influencing the Prognosis in Tuberculous Pleurisy.—It is a noteworthy fact, not appreciated to the extent it deserves, that when pleurisy is followed by tuberculosis, the outlook for the patient is not grave, as a rule. Thus, it has been noted for many years that pleurisy complicating active tuberculosis may be "beneficial"; it is often observed to arrest the tuberculous process in the lung, and the patient improves temporarily, or even recovers, provided, of course, that only tubercle bacilli and not pyogenic organisms are responsible for the inflammation. At one time it was suggested that these pleurisies act beneficially by compressing and immobilizing the affected lung, thus affording it rest and an opportunity for the lesion to cicatrize, as we aim when inducing pneumothorax for therapeutic purposes. But further observations has shown that the mechanical factor is by no means the main one. It has been noted that in cases in which the effusion is slight in amount, and only short in duration, the effect on the lung may prove very salutary. In fact, in many cases of dry pleurisy followed by, or complicating, phthisis, the tuberculous process is mild, sluggish in its progress, and shows strong tendencies to heal. Thus Königer[1] found among 49 cases of initial pleurisy, only 1 in whom the tuberculous process pursued a progressive course. Among 29 cases of secondary pleurisy complicating active tuberculosis, the disease was favorably influenced in 27 cases. It is noteworthy that during the course of initial pleurisy, observes Königer, "open" tuberculosis, with tubercle bacilli in the sputum, is extremely rare. Among 78 cases he could find only 1 of this type, though in many, extensive changes in the lung could be made out, and they expectorated considerable sputum. In my own experience also I recall but few cases of primary pleurisy in which tubercle bacilli were detected in the sputum microscopically. Furthermore, in patients with progressive and extensive tuberculous lesions in the lungs, with excavations which have

[1] Ztschr. f. Tuberk., 1911, **17**, 529.

rapidly formed, there is but rarely observed one who gives a history of pleurisy preceding the onset of phthisis. Of course, many adhesions may be found when these patients come to autopsy, but, as a rule, the pleural lesions had not manifested themselves by a reaction producing special symptoms. *Acute progressive phthisis following primary pleurisy is extremely rare*, excepting in acute miliary tuberculosis, or in acute pneumonic phthisis which, in rare instances, is accompanied, or masked, by a pleural effusion (see p. 505). In our daily practice we meet with cases of chronic tuberculosis manifesting itself mainly by a thick pleura, in addition to the infiltration or excavation of the apex, living on for many years. Many of these are told that "one lung is completely gone," yet they live on, and may even be fairly active at their avocations, despite the activity of the tuberculous process in the lungs and pleura. Among these are the cases with dextrocardia, sinistrocardia, immobility of the diaphragm, etc., all due to massive pleural adhesions, in whom the prognosis as regards duration of life is much better than in those in whom the pleura shows no signs of having been implicated materially in the tuberculous process.

The reasons for the salutary influence of pleurisy on the pulmonary tuberculous process are not definitely known. Only rarely is the mechanical factor instrumental because, as was stated above, dry pleurisy, as well as small effusions, often act in the same manner. The biochemical action of the exudate, or the inflammatory reaction of the pleura, may be the cause, as Königer suggests, but so far we have no proof for this contention. At any rate, it seems to me that the salutary effect of pleurisy on the pulmonary process is due to the tendency it has to induce a productive inflammation. Fibrosis appears to be Nature's weapon against the destructive action of the tubercle bacillus. Kaufmann[1] found that aseptic irritation of the pleura in dogs, *e. g.*, introduction of nitrogen while creating a pneumothorax, induces a proliferation of connective tissue not only in the pleural sheets, but also in the interstitial pulmonary tissues, etc. Other pathological processes characterized by fibrosis also have a good influence on tuberculosis, as is the case with gout, interstitial nephritis, some cases of tertiary syphilis, chronic alcoholism, etc. (see p. 598). As to the substance which is effective in producing a proliferation of connective tissue during an attack of pleurisy; whether it is biochemical, or some specific antibody, we are in the dark. It is a subject which deserves further investigation.

The prognosis in primary tuberculous pleurisy is thus not so gloomy as some would lead us to believe. The patient may be told that after the pleurisy has passed, his chances of developing phthisis are greater than in the average human being, still he is by no means invariably doomed. The majority pass through life without becoming phthisical. If he should be unfortunate and develop pulmonary tuberculosis, he

[1] Beitr. klin. d. Tuberk., 1912, **23**, 57.

may be told that his outlook is rather favorable. In most cases the disease pursues a mild, slow course and tends to recovery.

Influence of Age on the Prognosis.—The prognosis is also greatly influenced by the age of the patient. Pleurisy with effusion in children is not followed by pulmonary tuberculosis, as a rule. In some, bronchiectasis remains for life, but the lesion is not tuberculous. From Allard and Köster's statistics it appears that the prognosis after idiopathic pleurisy is much brighter in early than in middle life, and, while the subsequent incidence of tuberculosis is only 30 per cent when the pleurisy occurred between the ages of six and ten, it is as high as 60.4 per cent when the pleurisy has occurred between the ages of thirty-one and thirty-five years. At the high age of sixty-six to seventy idiopathic pleurisy is also followed by tuberculosis in 40 per cent of all cases. It appears also that in tuberculosis following pleurisy, when it does occur in children, the prognosis is better than when it occurs in adults. The tendencies to recovery are more pronounced in children than in adults.

Symptoms of Tuberculosis Following Pleurisy.—It is important to be able to single out the cases in which phthisis is likely to develop after an attack of pleurisy so as to institute timely treatment. We could then permit those who are unlikely to become phthisical to pursue their life-work without fear lest their occupation will be instrumental in promoting the onset of the disease. It is unfortunate that while this problem confronts us very frequently we are not always able to give definite information to the patients during the course of the pleural affection. In some, the pain in the chest, the fever, the cough, etc., disappear within a few days and we may be deceived by the prompt recovery. Within a few weeks, or months, fever, cough, expectoration, nightsweats, emaciation, etc., make their appearance, and signs of phthisis are discovered in the chest. In others, as I have observed in rare instances, the recovery is complete and the patient returns to work, but several months later, without any assignable exciting cause, symptoms of tuberculous meningitis appear, and kill him promptly. In many with effusions the fluid is absorbed within a few weeks, but the patient keeps on ailing, coughs, expectorates, has mild fever and nightsweats, and remains anemic and debilitated; signs of a tuberculous lesion in the lung may or may not be clearly evident.

A patient with any form of pleurisy who does not recover his general health and well-being soon after the fever abates, or the effusion is absorbed, should be considered as probably tuberculous and a careful search should be made for physical signs of a tuberculous lesion in one of the apices. It must be emphasized, however, that in these cases the tuberculous lung lesion is almost invariably localized in one of the apices. When physical examination of the chest shows signs of a thick pleura exclusively over the base, where the pleural friction was audible, or the exudate had occurred, the chances of the lesion being tuberculous are remote; these lesions usually turn out to be bronchiectatic

and not tuberculous. When signs of a thick pleura, such as impaired resonance, feeble breath sounds and moist consonating rales found exclusively at the base, are due to tuberculosis, there is also to be made out a tuberculous lesion in the apex in nearly all cases. In doubtful cases of this sort roentgenography may be of immense value in localizing an apical lesion. Of course, the sputum is to be examined repeatedly for tubercle bacilli.

Patients who recover promptly after an attack of pleurisy may be pronounced free from active tuberculosis at the time. But, as was already shown, they are more likely to develop phthisis in later years. It may be stated as a general rule that this predisposition wanes with the advance of time after the attack of pleurisy. Allard and Köster found from their extensive statistics that in the majority of cases which became tuberculous, to be precise in 85 per cent, the tuberculous process flared up within five years after the attack of pleurisy. In younger individuals, however, it appears that pleurisy is followed by pulmonary tuberculosis much later.

These conditions are illustrated even more drastically by the American Medico-Actuarial Investigations of 1914. It was found among insured lives the incidence of deaths due to tuberculosis was as follows:

		Actual deaths.	Expected deaths.	Ratio.
A.	One attack within 2 years of application . .	228	154.8	147
B.	" " " 2 to 5 years of application	262	179.7	146
C.	" " " 5 to 10 " "	253	223.7	113
D.	" " more than 10 " "	528	573.7	92

In group A and B the death rate from phthisis was observed to be about three times the normal; in C about twice the normal, and in D it was about normal, indicating that the danger of phthisis decreases with the time that passes after an attack of pleurisy.

From these figures, as well as from daily observation, it appears that if the patient has completely recovered his health after an attack of pleurisy he should be told that while he may reëngage in his vocation, he must be careful in his mode of life during the ensuing five years.

A careful inquiry should be made into the past history of the patient. Many with so-called primary pleurisy have, in fact, presented symptoms of phthisis for months before the appearance of the pain in the chest or the symptoms and signs of an effusion, but they disregarded them, as has already been emphasized. In such cases the diagnosis of tuberculosis may be safely made. It is in these cases that tubercle bacilli are frequently found in the sputum.

Prognosis in Secondary Pleurisy.—In pleurisy developing during the course of pronounced phthisis the outlook depends mainly on the underlying disease, on the condition of the tuberculous lungs, as well as on the resisting powers of the patient. In unilateral tuberculous lesions, which show no tendency to progression, an attack of dry pleurisy may have no effect on the ultimate outlook. It is likely to torture

the patient by the pain in the chest and shoulder that it inflicts, and its likelihood to recurrence; but the prognosis as regards the duration of the disease may even be improved, as has already been mentioned. The same is true of an effusion. In some cases I observed that it has been the turning-point for the better when, before the onset of the complication, the course of the lung disease was active and progressive. The effusion may be slow in disappearing, but when it is finally absorbed the patient feels well, even though he remains with a thick pleura and with signs of adhesions producing dyspnea on exertion.

It is different with extensive bilateral lesions. While in some cases we may even here note improvement, in the majority the reverse is true. The effusion is likely to aggravate the cough, produce distressing dyspnea, and the fever rises higher. Hectic fever is not uncommon. At times the end comes suddenly through asystole, but in most cases the course is lingering. Repeated tappings are of little or no avail in many cases. In fact, it has been my impression that, very often, the prognosis is distinctly aggravated by tapping the exudate, excepting when the effusion is very copious and produces menacing symptoms through its mechanical effects.

Prognosis in Empyema.—Empyema is one of the most dangerous complications of phthisis. Spontaneous absorption hardly ever occurs. Operations for the removal of pus are very unsatisfactory. The result is usually that the fever, cachexia, and amyloid degeneration of the viscera carry off the patient sooner or later. I have seen a few cases of empyema in which the pus found its way into a bronchus and was expectorated. The patients were "cured" of the empyema, but the tuberculous process proceeded on its course to a fatal termination.

In general it may be stated that the vast majority of empyemata in tuberculous subjects are in truth cases of pyopneumothorax, and the prognosis is the same as in the latter condition (see p. 533).

In very rare instances the pus becomes encapsulated and while remaining within the chest gives no more trouble. I have discovered such pus pockets in individuals who felt well and worked for years. Schweizer[1] reports such cases of *chronic empyema* lasting for twelve to eighteen years and not interfering with the patient's earning capacity. Recently such a case was referred to me with a view of deciding whether there is an active tuberculous lesion in the lung. However, these patients show no symptoms or signs of active tuberculous lesions, as a rule.

Another mode of termination of empyema remains yet to be mentioned. While in rare cases repeated tapping may finally clear the pleura of the purulent exudate, in still rarer instances it has been observed that the pus is spontaneously absorbed and the patient remains with a thick pleura, pulmonary retraction, dilatation of the bronchi, and deformities of the chest and spine. In very rare instances

[1] Corr.-Bl. f. Schweizer Aerzte, 1919, **49**, 385.

the abscess in the pleural cavity becomes encapsulated and the patient may go around for many years without considerable inconvenience. The pus in these cases changes in appearance, becoming milky-white, or ivory in color, as in chylothorax, and is in fact converted into cholestrin. In one case, with a history of pleurisy twelve years before he came under my observation, I withdrew with an exploratory syringe yellowish-white fluid, which, on microscopic examination, showed an abundance of cholesterin crystals. In another case the woman, forty-five years of age, had a very pronounced kyphoscoliosis, the result of an empyema during childhood. Finding signs suggestive of a pleural effusion, I inserted an exploratory needle and withdrew milky fluid which microscopically was found studded with cholesterin crystals.

CHAPTER XXVII.

PNEUMOTHORAX.

THIS is the most frightful complication of pulmonary tuberculosis. It is of more significance than copious pulmonary hemorrhage because the latter only terrifies the patient, and its ultimate prognosis is usually favorable, as we have already shown, but pneumothorax is deadly, and the victim is justified in his apprehension that the collapse, and agonizing dyspnea, are indications that he is breathing his last. From West's statistics it appears that 70 per cent of patients attacked by pneumothorax die, and in phthisis the proportion is even higher.

The frequency of pleural adhesions in patients with pulmonary phthisis explains why all suffering from this disease do not develop pneumothorax. Eugene L. Opie,[1] making autopsies, found that nearly half of the focal tuberculous lesions are situated immediately below the pleural surface. It is not uncommon to find a calcified nodule immediately below the puckered pleura and about it upon the adjacent pleura a group of small nodules. Fibrous adhesions usually bind together the adjacent pleural surfaces. It is thus clear that if there were no adhesions, pneumothorax would be the most common complication of pulmonary tuberculosis.

Spontaneous Pneumothorax.—Many authors have applied this term to cases in which rupture of the pleura and entry of air into its cavity have occurred in an apparently healthy individual who has not known of any premonitory symptoms, nor of an acute onset, and who develops no subsequent hydrothorax or pyothorax. Indeed, I have had patients coming into my office complaining of breathlessness and an examination disclosed the presence of pulmonary collapse and air in the pleura. In most of these patients the air is absorbed within three to six weeks. In three cases under my care effusions developed, but they remained sterile and were absorbed spontaneously. Nikolski[2] collected from literature 66 cases of this kind and he found that 59 recovered completely within eight weeks and 3 within four months. But there have been reported chronic and persistent cases. Bittorf[3] mentions a case lasting twenty-five years and Whitney[4] one of seven years' duration.

The origin of this form of pneumothorax has been discussed by many medical writers. The consensus of opinion appears to be that they

[1] Jour. Exper. Med., 1917, **25**, 855.
[2] Ueber den spontänen Pneumothorax, Inaug. Dis. Giessen, 1912.
[3] München. med. Wchnschr., 1908, **55**, 2274.
[4] Philadelphia Med. and Surg. Jour., 1886, **115**, 397.

are due to tuberculous lesions of the lungs or pleura. Hamman[1] agrees with those who hold that the commonest cause is a pleural adhesion of a tuberculous character tugging upon the visceral pleura and producing a rent. The fact that no infection of the pleura occurs shows that the rent occurs about the adhesions and not over the seat of the parenchymatous lesion. Indeed, Flint, Letulle, and West have reported cases in which only a single subpleural tuberculous nodule was found to have ruptured and permitted air to enter the pleural cavity. That the rent need not be very large to produce this effect is evident from the fact that surgeons, while anesthetizing the brachial plexus with cocaine, have produced pneumothorax through a puncture with the hypodermic needle. Schepelmann and A. Vischer have reported such cases. Similarly, pneumothorax is in rare instances produced while performing paracentesis for exploratory purposes.

While in the vast majority of cases the occurrence of this form of pneumothorax is an indication that there is a pulmonary tuberculous lesion, there are exceptions. There is considerable evidence that spontaneous pneumothorax may occur as a result of rupture of emphysematous blebs or bullæ. Zahn,[2] Bach,[3] and many others have described such cases, and they speak of pneumothorax due to pleural rupture without inflammation. A. Cahn[4] believes that sudden increase in the intrapulmonary pressure may be effective in tugging any small adhesion and thus lacerate the visceral sheet. Schule[5] reports a case in which violent laughter was the exciting cause of a pneumothorax. It may also be caused by rupture of interstitial emphysematous blebs, the air entering the interstitial tissues and reaching the visceral pleura, forming a vesicle which ruptures. We often see this mechanism in cases in which therapeutic pneumothorax has been induced. During the late epidemic of influenza I met with a case which could only thus be explained. Spontaneous pneumothorax also occurs in rare instances in gangrene of the lung, bronchopneumonia complicating influenza, etc.

Spontaneous pneumothorax occurs more frequently in males than in females. Nikolski found it in 75 males to 14 females; Fussell and Riesman,[6] 45 and 10, respectively. Overexertion, cough, etc., are said to be the usual exciting causes, but at times no cause can be discovered.

In most of the patients the air in the pleura is absorbed within a shorter or longer time, and they recover completely. In some, the recovery is but temporary, and within a few months or years there appear symptoms and signs of a pulmonary tuberculous lesion. In rare cases there have been observed *recurrent attacks of spontaneous pneumothorax*. Gabb, Vitvitski, Sale, Clyde L. Cummer,[7] and others reported such cases. In one case eleven recurrent attacks were

[1] Am. Jour. Med. Sci., 1916, **151**, 229.
[2] Virchows Archiv. 1891, **122**, 197.
[3] Brauer's Beiträge, 1910, **18**, 21.
[4] Deut. med. Wchnschr., 1917, **43**, 1469.
[5] Ibid., 1918, **44**, 10.
[6] Am. Jour. Med. Sci., 1902, **134**, 218.
[7] Ibid., 1915, **150**, 222.

observed. On the whole the prognosis is very good indeed. In fact, in 4 cases under my care, there was a history of indefinite symptoms of pulmonary tuberculosis for some time before the occurrence of pneumothorax. But the collapse of the lung was apparently of the kind called "providential" by some writers. After the air in the pleura was absorbed, the patient felt well. One has thus kept well for ten years.

Tuberculous Pneumothorax.—As a complication of phthisis, pneumothorax is of graver significance than when occurring in apparently healthy individuals. The frequency of this complication varies with the character of the clinical material. It is not very frequent in hospitals for advanced cases because only patients with old lesions, in whom pleural adhesions prevent its occurrence, are admitted. According to Powell, about 6 per cent of fatal cases of phthisis at the Brompton Hospital at London succumbed with pneumothorax; Williams found 10 per cent, and Weil even 13 per cent. On the other hand, Biach, among 715 tuberculous cases, found only 0.73 per cent complicating pneumothorax; Blumberg, among 425 cases, 3.1 per cent; Drasche, among 26,231 cases, 1.46 per cent. At the Montefiore Hospital it constitutes about 3 per cent of the fatal cases.

As was just stated, these wide differences in the proportion of complicating pneumothorax are to be ascribed to the differences in the material. In many hospitals for consumptives we meet with cases of sudden death during the night. Some of these are due to sudden profuse internal hemorrhages, but in most cases the cause is pneumothorax, which killed the patients before aid could be summoned.

The lesion is more likely to occur in the left than in the right pleura. From a collection of 234 cases reported by Louis, Walshe, West, and himself, Powell finds that in 95 the rent was in the right and in 139 in the left pleura. He attributes it to the greater frequency with which the left lung becomes the seat of tuberculous disease.

Symptoms.—The onset is sudden, unexpected. The patient has known that he is tuberculous for some time, and may have been assured that his prospects for ultimate recovery are good. But suddenly, like a thunderbolt out of a clear sky, after a fit of coughing, some slight exertion, or without any exciting cause at all, he is seized with a sharp agonizing pain in the chest, he feels as if "something has given way," or as if something cold is trickling down his side. He at once sits up in bed holding his hand fast over the affected side, gasping for breath. Acute distressing dyspnea, cyanosis, a small, rapid and feeble pulse, cold, clammy extremities and other phenomena of collapse soon make their appearance. The facial expression is that of profound agony, the eyes prominent, the lips livid, and the forehead clammy. The respirations are frequent—fifty or more per minute, and superficial. The temperature, which may have been elevated for some time, suddenly drops to below normal and the cough, which may have been annoying before the accident occurred, ceases for a time; perhaps because of the pain the patient restrains himself.

In very acute cases the patient may expire within a few hours as a result of profound shock, dyspnea, and heart failure. Many of the cases of sudden death in phthisis are due to this cause. But in most cases the circulation adapts itself by degrees to the altered conditions of the thoracic viscera, the dyspnea is ameliorated, the temperature rises to above normal, and the patient feels somewhat relieved, the air-hunger not being so acute as at the onset, though he still breathes forty or more times per minute, and is still cyanosed. Within a few days, usually between the third and fifteenth day, an effusion of fluid into the affected pleura is found, hydropneumothorax, or pyopneumothorax.

The size of the perforation into the lung has but little influence on the acuity of the distress—a small opening the size of a pinhead may permit the entry of sufficient air into the pleura to collapse the lung completely and to displace the thoracic and abdominal organs just as well as a larger one. In fact, in some quickly fatal cases only a small opening, or slit, is found at autopsy, while in others, with large openings, little distress is seen, healing is rapid, and the patient may last for months. At the necropsy it is found that the opening is usually small, linear, slit-like, and occasionally circular, at times attaining the size of a dime. In some cases there are two or even three perforations.

Mechanism of Pneumothorax.—It is of clinical significance whether the perforation closes speedily and no more air or pus can pass into the pleural cavity, thus allowing absorption of the air. The symptoms, prognosis, and treatment depend mainly on this point. There are described in text-books three varieties of pneumothorax—*open*, *closed*, and *valvular*. In the open variety there is a patent opening which permits air to pass in and out of the pleural cavity, and the tension within the affected pleura is equal to that of the external air. In the closed variety the perforation has healed, and the air in the pleural cavity may be absorbed sooner or later, as is the case with induced therapeutic pneumothorax, with or without the development of an effusion which is generally serous. In the valvular variety, during inspiration or cough air enters freely into the pleura, but is prevented from coming out again during expiration by a valve or contraction of the slit. The result is that the tension within the pleural cavity becomes very high and, pushing the mediastinum to the opposite unaffected side, causes distressing dyspnea, cyanosis, and heart failure, till the patient is no longer able to cope with the situation and succumbs.

This interpretation of pneumothorax has been questioned by West, Bard, C. P. Emerson, Castaigne, and others. West says: "All pneumothorax is at first valvular, at any rate more or less, *i. e.*, the air finds more or less difficulty on expiration. Thus the pleura becomes more and more full of air and the lungs more and more compressed, and this obviously tends to close the hole more or less completely. When the hole is of ordinary size, it will become patent on inspiration and thus admit air, but only so long as the pressure in the pleura is less than

that of the air in the air tubes. As soon as the pressure on the two sides is equal no more air can enter, and the hole remains closed. If the edges cohere, the hole will remain permanently closed; if not, as soon as the pressure in the pleura is diminished, as it may be by paracentesis, the orifice may open again into the pleura. This is the explanation in many cases of the return of dyspnea after paracentesis."

It should also be mentioned that the acute and distressing symptoms observed in pneumothorax are not necessarily due to high pressure within the pleura. It has been stated that when the intrapleural pressure reaches 6 to 10 cm. of water, dangerous symptoms are bound to ensue. But that this is not a fact has been learned recently through experiences with therapeutic pneumothorax. Much higher pressure within the pleural cavity is often produced, over 20 cm. of water, without producing acute and menacing symptoms. Actual measurements have shown that with a pressure of 15, 18, 25, and Bernard even raised its exceptionally to 35 or 45 cm. of water, the only effect was that the mediastinum was displaced to the opposite side, but the circulation adapted itself, and the patient felt quite comfortable, at least during rest. The writer has repeatedly observed that when fluid appears in an artificial pneumothorax, the pressure within the pleural cavity rose to 25 or 30 cm. of water, yet the distressing and menacing dyspnea of spontaneous pneumothorax was lacking.

The accommodative powers of the pleural cavity have been studied by Emerson.[1] He found that the chest, by elevation of the ribs and descent of the diaphragm, can accommodate various quantities of fluid without any change of pressure. If fluid is continuously injected into the pleural space, the pressure, of course, must rise, but it tends to do so in stages or jerks, owing to attempts on the part of the chest to accommodate itself to the increase and so keep down the pressure. He ascribes this to a special reflex mechanism.

As has been pointed out by Sir R. Douglass Powell, the displacement of the mediastinum is not necessarily due to the pressure exerted by the air in the pleural cavity. His manometric measurements have revealed no positive pressure in pneumothorax. From his investigations he is inclined to believe that the dislocation of the heart is due to the unopposed traction exerted by the elastic unaffected lung. Because they are no longer held up by the elasticity of the lung, the diaphragm and the abdominal viscera sink downward.

Clinically this view is confirmed by the fact that tapping a pneumothorax is not always effective in relieving the patient for any duration of time. In fact, better results are now obtained by, instead of tapping the pleura, insufflation of more gas (see p. 859). The recent experimental investigations of Evarts A. Graham and Richard D. Bell[2] of the Empyema Commission of the United States Army tend to confirm this view of the mechanism of pneumothorax.

[1] Johns Hopkins Hosp. Rep., 1903, 11: 1.
[2] Am. Jour. Med. Sci., 1918, **156**, 839.

Partial Pneumothorax.—In old chronic cases of phthisis we meet with partial pneumothorax in which there is a perforation into the pleural cavity, but owing to dense adhesions the air is only filling up a limited pouch, at a place where the pleural sheets are not adherent. The onset is less acute and the symptoms of collapse are usually absent. The patient may have some pain in the chest, dyspnea, etc., but these attract little attention in a disease like phthisis in which these symptoms are so frequent without the occurrence of pneumothorax. Careful physical examination may disclose signs of the condition, but it is easier to find it out with the aid of roentgenography. The writer[1] has reported cases in which roentgenography could not decide. It is often mistaken for a large cavity, especially when it is localized over an apex, but even in the lower parts of the chest it may exquisitely simulate pulmonary excavation.

Latent Pneumothorax.—At times we meet in tuberculous patients pneumothorax without a history of an acute onset with pain, dyspnea, collapse, etc. In some of these cases careful inquiry elicits a history pointing to a subacute onset, but such symptoms are quite common in chronic phthisis without this complication. In one case admitted to the hospital we found complete collapse of the lung and we at first suspected an artificial pneumothorax, produced before admission, but it turned out to be a latent case. Several cases of this type have presented themselves for examination at my office. No history of onset could be elicited, yet the pleural cavity was full of air and the lung collapsed.

In chronic phthisis we also meet with cases in which there is a sudden onset with all the symptoms of this accident, but physical examination fails to reveal any of the signs. The French call it *pneumothorax muet*, the mute form. In these cases the signs do appear, however, within a few days. In one of my cases of this character a roentgenographic plate showed that the air was filling the thoracic cavity for an inch or two along the axillary line. In others there was an interlobar air pouch. These forms are best diagnosticated with the roentgen-rays.

Double Pneumothorax.—Double pneumothorax has been met with in phthisis on exceedingly rare occasions. On Plate XXIII is shown the roentgenogram of a case that came under the writer's observation. It is incompatible with life. But D. Hellin,[2] R. Staehelin,[3] and E. A. Gray[4] mention cases which lasted for days.

Physical Signs.—The affected side of the chest is larger—in the maximum inspiratory position; the shoulder raised, the intercostal spaces obliterated, tense and tender to the touch. While the number of respirations is fifty or more per minute, movements of the thorax

[1] Arch. Int. Med., 1917, **20**, 739.
[2] Mitt. a. d. Grenzgeb. d. Med. u. Chir., 1907, **17**, 414.
[3] Mohr and Staehelin's Handbuch der inneren Medizin, Berlin, 1914, **2**, 756.
[4] Illinois, Med. Jour., 1919, **35**, 252.

are seen only in the unaffected side, while the affected side is fixed, almost immobile. In the vast majority of cases the apex beat cannot be seen, but when visible it is found at the left axillary line in right-sided pneumothorax and at the xyphoid cartilage, or even beyond it, in left-sided perforations. Vocal tremitus is abolished over the affected side.

Instead of the dull note which was found before the accident, the affected side emits a hyperresonant, sometimes a tympanitic note, depending on the tension of the air within the pleural cavity. By comparison, the unaffected side appears to emit a defective or dull note. In cases in which the upper part of the pleura is adherent, and does not collapse, the apex is dull or "boxy" on percussion. When later fluid makes its appearance in the pleural cavity, we elicit a flat note over the lower part of the chest and the flatness changes its level with a change in the patient's position (see Figs. 1, 2, and 3, Plate XXIII). *Shifting dulness is pathognomonic of air and fluid in the pleural cavity.* Displacement of the thoracic and abdominal viscera can be made out more or less easily by percussion. In right-sided lesions the liver dulness disappears altogether, or is displaced downward, and the heart is shifted to the left, even as far as the axillary line; in left-sided pneumothorax the heart dulness may be completely absent, or displaced to the right, and the splenic dulness may also be absent. In fact, the spleen and the liver may be felt distinctly low in the abdomen. The displacement of the heart may be noted a few minutes after the occurrence of the accident.

We may also elicit various metallic or amphoric notes on percussion, especially with a coin placed over the chest and tapping it with a stick or pencil, while listening with the naked ear or stethoscope over the opposite side of the chest. A thimble over the middle finger may be used percussing over the nail of the ring finger placed on the chest wall. The metallic sound heard while listening on the chest is exquisite. This method has the advantage that no assistance is needed to bring out the so-called coin, or penny sound. Biermer's and Wintrich's signs, as well as cracked-pot resonance, may be elicited in many cases.

Auscultation shows complete absence of breath sounds over the affected side of the chest in cases in which the opening is small or closed and the lung is completely collapsed. When the upper parts of the pleura are adherent, the auscultatory phenomena of the original lung lesion are audible, but below no sounds at all are heard. But in most cases there are heard amphoric breath sounds at some point between the shoulder-blades. Exceptionally we meet with a case of pneumothorax in which the voice and breath sounds are audible in an exaggerated form all over the affected side. When the opening into the lung is large, permitting the passage of air from the bronchi into the pleural cavity, we may hear an exquisite variety of amphoric breathing, or metallic sounds, which are characteristic. The voice sounds, as well as the cough, may also have a metallic echo.

The *splashing* or *succussion sound* is audible at a distance in many cases, and the patients themselves are annoyed by it. Some patients know how to jerk their bodies to produce it to the best advantage. I have had patients in whom the succussion sound was the only indication of fluid in the thorax, all other signs being absent because of the depression of the diaphragm, the result of the pressure exerted by the tension of the air in the pleura. It is an excellent proof of the existence of air and fluid in the pleura. It is stated that it may be elicited in the stomach and colon, but I have not met with a case in which this vitiated a diagnosis.

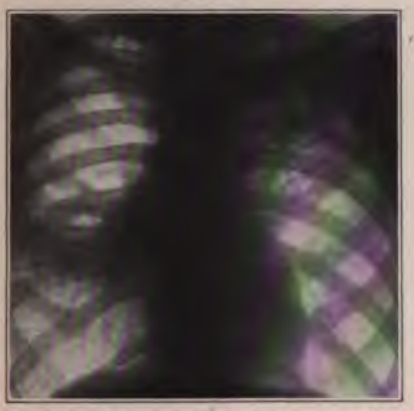
Metallic Tinkling.—A clear muscial note, heard at intervals on listening over a hydropneumothorax, resembling a drop of water falling into a reverberating vessel, may be heard in some cases. At times it is only heard after cough. It is apparently not due to the falling of a drop at all, but to a rale produced in some portion of the lung which acquires a metallic character by reverberation or air bubbles exploding in the fluid.

Diagnosis.—The diagnosis of pneumothorax has undergone quite some changes within recent years since we have had an opportunity to study this condition produced artificially in tuberculous patients for therapeutic purposes, and also since we employ roentgenography diagnostically in chest conditions. We now have explanations for some phenomena which were formerly obscure, and we know that certain signs formerly considered pathognomonic of pneumothorax, are not at all invariable accompaniments of the disease.

In the usual case of pneumothorax during the course of phthisis the sudden onset of urgent dyspnea, pain in the chest, collapse, etc., coupled with physical signs of pulmonary collapse, suffice to establish the diagnosis. But there are many sources of error. We may have pneumothorax without any of these acute symptoms, as has been already stated. In fact, since the *x*-rays have been employed the number of latent and mute cases of pneumothorax has enormously increased. On the other hand, we meet in phthisis cases of acute dyspnea, pain, and even collapse not due to this accident. In several instances paroxysmal tachycardia in hysterical female patients has simulated pneumothorax to a marked degree. Especially difficult are the cases of localized pneumothorax, because the mediastinum is not displaced, and it may even be drawn to the affected side; a thickened pleura may obscure the tympany, and the absent, or amphoric, breath sounds may be otherwise interpreted. At times it is very difficult to differentiate a partial pneumothorax from a large pulmonary cavity, and before the advent of roentgenography mistakes of this kind were more frequently made than at present. The differentiation is usually of practical value, because the prognosis in cases with large excavations is very unfavorable, while with a localized pneumothorax it is more hopeful.

PLATE XX

FIG. 1



Localized pneumothorax in upper third of right side. Interlobar fissure markedly thickened. Extensive tuberculous changes in upper lobe of the left lung.

FIG. 2

Localized pneumothorax in right side of thorax. Note the thick bands of adhesions running from the diaphragm and mediastinum. Diaphragm elevated.

FIG. 3

"Ballooning." Pneumothorax at high pressure in the left pleura. Hernia of the left pleura at the right side of the sternum.

FIG. 4

"Ballooning." Pneumothorax in left pleura. Lung not completely collapsed. Left pleura protruding into the right side of the chest.

PLATE XXI

FIG. 1

Dense infiltration of the upper half of the left lung with displacement of the heart to the left. Right lung emphysematous.

FIG. 2

From same patient as Fig. 1. Spontaneous pneumothorax, air filling left pleural cavity and displacing the heart to the right.

FIG. 3

Pneumothorax in right pleura extending in a thin layer of air from the diaphragm to the apex. Right lung slightly collapsed and presents consolidation at its lower third. The rest appears studded with cavities and calcified nodules. Lower half of left lung emphysematous; upper half nodular infiltration, especially at axilla. Heart and trachea displaced to the right.

FIG. 4

Hydropneumothorax in the right pleura.

PLATE XXII

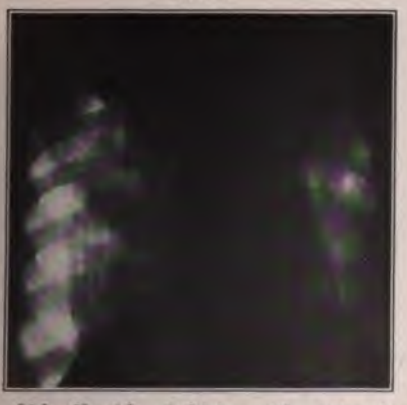
FIG. 1

Left pleura filled with air, but large cavity with dense walls under second and third interspace did not collapse. Nodular infiltrations throughout right lung. Dilated bronchi and enlarged glands in hilus region.

FIG. 2

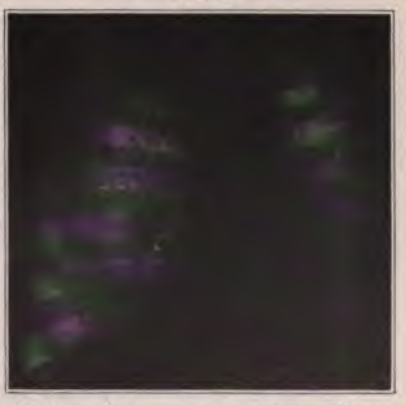
Complete pneumothorax in left side. Note the left lung compressed against the mediastinum which is markedly displaced to the right.

FIG. 3



Old fibroid phthisis with extensive involvement of the left lung and pleura. Spontaneous pneumothorax in right pleura.

FIG. 4



Diffuse tuberculous process all over both lungs, superadded on an anthracotic lung; marked peribronchial infiltrations and calcified glands along the hilus. The apex is infiltrated and adherent in the left side; below the clavicle there is a circumscribed pneumothorax, which on physical exploration gave signs of a cavity. The lower half of the left pleura is thickened, which cannot be differentiated in the roentgenogram from fluid.

PLATE XXIII

FIG. 1

Hydropneumothorax in left pleura. Patient in erect position.

FIG. 2

Same patient as in Fig. 1, but body leaning to the right.

FIG. 3

Hydropneumothorax. Patient lying on the side.

FIG. 4

Double pneumothorax.

Illustrating Shifting Fluid in Hydropneumothorax.

Even in cases with complete collapse of the lung, tympany may not be elicited on percussion, as we have learned lately in cases of artificial pneumothorax. It appears that it all depends on the tension of the air within the pleural cavity. In hydropneumothorax, tympany is found when there is but little fluid and considerable air; but when the effusion is copious we get flatness which disappears when the fluid is aspirated, provided the pleura is not too thick.

The position of the heart is usually of assistance in deciding whether we deal with a large cavity or a pneumothorax: In the former it is displaced toward the affected side, while in the latter it is moved away from it. But even here there are many important exceptions, owing to previous pleural adhesions, etc. Roentgenography usually decides, but not always.

The signs obtained on auscultation differ very much in cases of open, as compared with closed pneumothorax, and in the latter cases it depends on whether the lung is completely or only partially collapsed. A closed pneumothorax with complete collapse is mute; no breath sounds at all are audible, as a rule. At times we perceive some bronchial breathing in the interscapular space emitted from the bronchi near the spine. In the open variety we usually hear amphoric breathing of an exquisite type. In many cases of phthisis, in which the pleura is free all over, it is adherent at its upper third, over the site of the main lesion, and does not collapse at that place, and we obtain the breath sounds and rales peculiar to the diseased lung.

The breath sounds often audible over a completely collapsed lung were formerly attributed to some opening into a bronchus, allowing air to pass in and out of the pleura. We now know that this is not always the case because in artificial pneumothorax, where an opening into the lung is positively excluded, we often perceive the same acoustic phenomena. It seems that the air in the pleural cavity is capable of transmitting the sounds in the bronchi when in a certain condition of tension. More often bands of adhesions running from the collapsed lung toward the periphery act as conductors of the sounds originating in the trachea and bronchi.

The *bell sound* is very frequently elicited in cases in which the effusion is not too thick, as in some cases of pyopneumothorax. It is easily elicited by placing a coin over the anterior surface of the thorax and percussing it with another while auscultating posteriorly or in the axilla. A clear, ringing, bell-like sound, which is characteristic, is heard. But exceptionally it is also heard over large cavities, or even a dilated stomach. It is often absent in pneumothorax; but when heard it is of significance, showing, as it does, air and fluid in the pleural cavity. We may hear it only with the patient in the horizontal position. In some it appears only after some of the fluid has been aspirated.

A positive diagnosis of pneumothorax can be made when one is alert and looks for it in every suspicious case. In most cases the

abrupt onset of the urgent symptoms and the physical signs suffice. In doubtful cases the roentgen rays decide easily and speedily.

A rare complication of pneumothorax, the spontaneous as well as the artificial varieties, is pneumopericardium—air entering the pericardial sac. We then have instead of the cardiac dulness, hyperresonance or tympany, sometimes cracked-pot sound. On auscultation we hear that the heart sounds are extraordinarily intensified, and a splashing sound is audible, or a succussion sound, synchronous with the systole. In the case observed by the author there was also a metallic tinkle and a friction fremitus, especially when the patient bent his body forward. Similar cases have been reported by Wenckebach,[1] Cowan,[2] Harrington and Riddell, and Meyer.[3] With the aid of roentgenography the diagnosis offers no difficulty.

Roentgenography.—The *x*-rays have their greatest field of usefulness in our attempts at discerning the changes in the thoracic organs when the lung is collapsed by air in the pleural cavity, especially in the localized variety of pneumothorax, which formerly escaped attention in most instances. Complete pneumothorax is clearly seen on the fluoroscopic screen or the roentgenographic plate. The affected hemothorax shows a very bright area, lacking in lung markings; in contrast with the opposite expanded lung it may be said to be brilliant. The ribs on the affected side are more sharply demarcated than on the opposite side; the costophrenic angle is clearly seen and runs rather acutely downward. The collapsed lung is seen lying near the mediastinum, or against the spinal column, as a dark band (see Figs. 1, and 2, Plate XXII). During respiratory efforts the collapsed lung becomes somewhat larger and brighter. In many cases of tuberculous pneumothorax some part of the lungs, especially the apex, is retained in its position by adhesions.

The mediastinal organs are, in most instances, displaced toward the unaffected side; very frequently the trachea is thus displaced. In many cases we may note rhythmic movements of the mediastinum, especially the heart; during inspiration the mediastinum is moved toward the unaffected side. The dome of the diaphragm is lower than that of the opposite side; its convexity is gone, and an almost straight line may be made out running downward and outward. The intercostal spaces are wider, and the ribs, as well as the diaphragm, move less during respiration than those of the unaffected side. In some there is complete immobility of the affected half of the thorax. In some cases the paradoxical phenomenon in the diaphragmatic motion, first described in Kienböck, may be noted. Instead of the normal, simultaneous contraction of the two halves of the diaphragm during each inspiration when they descend like pistons, there is observed a dissociation of this movement. The two sides of the diaphragm behave like two sides of a balance: While the unaffected half descends the affected half rises into the thoracic cavity.

[1] Ztschr. f. klin. Med., 1910, **71**, 402.
[2] Quarterly Jour. Med., 1914, **7**, 165.
[3] Medical Record, 1915, **88**, 991.

When fluid appears in a pneumothorax it is easily discerned with the *x*-rays. It is, however, important that the examination should be made with the patient in the erect posture, otherwise the fluid may spread out all over the chest and thus escape notice by those who have little experience with these cases. The fluid sinks down, and is seen as a deep shadow occupying the lower part of the chest, while the upper part, just above the level of the fluid, is bright. It has been well compared with a bottle half-filled with ink. The line of demarcation between the fluid below and the air above is sharply drawn, which is in contrast with effusions in pleurisy in which the dark shadow of the fluid merges by degrees into the bright lung tissue above. Inclining the patient to the side will shift the level of the fluid (see Figs. 1, 2, and 3, Plate XXIII). In some cases shaking the patient may cause agitation of the fluid within the chest, showing the mechanism of succussion.

Localized Pneumothorax.—As has already been stated, the diagnosis of localized pneumothorax is at times important owing to the difference in the outlook for the patient: The prognosis is good in many cases of localized pneumothorax, while it is poor in those with large pulmonary excavations. In our attempts at this differentiation we should bear in mind the following points:

The history of the onset is most important in nearly all doubtful cases. A pulmonary cavity of large dimensions does not appear suddenly, while signs of a localized pneumothorax appear within a few minutes. If we have had the patient under observation for some time, the sudden appearance of signs of excavation, such as tympany, amphoric breathing, and pectoriloquy over a circumscribed area, should suggest pneumothorax. In most patients we find that there has been a sudden change for the worse, even in such as have been doing quite well. A sharp, stabbing pain in one side of the chest is felt, followed by dyspnea, cyanosis, prostration of variable degrees, etc. But we meet with many cases in which the history is negative.

While in some cases a localized pneumothorax may prove fatal within a short time, in most the acute symptoms abate within a few days, the pain disappears, the dyspnea is ameliorated, though the patient remains short-winded on slight exertion.

Physical exploration of the chest shows that in localized pneumothorax the cavity is, as a rule, "dry," no adventitious sounds are audible; while large excavations usually show large, moist, consonating rales and gurgles. A large, "dry" cavity, especially when extending to the axilla, should not be accepted as such without careful investigation. The breath sounds in pneumothorax are distinctly amphoric or metallic; such exquisite metallic sounds are exceedingly rare in cavities. In the former a metallic tinkle may be heard, which is exceedingly rare in the latter. In cavities bronchophony is the rule, and whispered pectoriloquy is frequently absent, while in localized pneumothorax the latter is commonly present and is strikingly pronounced, clear, and articulate, usually perceived as if spoken directly into the stethoscope, a phe-

nomenon exceedingly rare in pulmonary excavations, in which only the spoken voice is transmitted. The whispered echo is also more frequently heard in pneumothorax. Moreover, in localized pneumothorax, especially the interlobar variety, whispered pectoriloquy is distinctly or exclusively heard high up in the axilla, which is very rare in cases with excavations. On inspection retraction of the chest wall is characteristic of large cavities, while bulging may be found, though rarely, in cases of localized pneumothorax. The location of the mediastinal organs gives no reliable criteria as to the condition. They are almost invariably displaced toward the affected side in large cavities but the adhesions which are instrumental in localizing the pneumothorax may also keep these organs in the place they had been before the rent in the pleura had occurred.

The roentgenographic findings are invaluable in most doubtful cases. A bright, circumscribed area, lacking in lung markings, when not surrounded by a thick, dark shadow, is almost pathognomonic of a localized pneumothorax. But at times even this is deceptive. The air pouch may be located anteriorly, while posteriorly adherent lung tissue screens it, and no bright area appears on the roentgenogram, as I have seen in some cases. On the other hand, the walls of the pulmonary cavity may not cast a shadow on the roentgenogram, and as a result we may find on the plate a picture clearly showing a pneumothorax, while the real lesion is a large pulmonary cavity. Such anomalous findings at necropsy have been reported by many clinicians and roentgenographers. The writer[1] has reported cases showing localized pneumothorax distinctly on the *x*-ray plate, yet the autopsy showed a large cavity in the lung. It seems to me that in such doubtful cases fluoroscopy is of more value than roentgenography. In localized pneumothorax we often see the mediastinum rhythmically moving during the respiratory act; during inspiration it is moved toward the affected side. This is best seen in artificial pneumothorax, after the first one or two fillings, when there is but a small air pouch in the pleura. In the spontaneous variety, when the adhesions are not dense enough to hold the mediastinum very fast, we may observe the same phenomenon, and this is never seen in cases of large cavities. In most cases it is, however, easy to differentiate on the roentgenogram between cavities and localized pneumothorax. In extensive disease, pulmonary cavities are usually multiple; they contain not only air, but also secretions which are not constant in quantity, changing intermittently, and bridges made up of connective tissue and bloodvessels. No clear, bright area lacking in lung markings is, as a rule, produced on the roentgenogram; their margins are more opaque and the pulmonary tissue around them is denser than in localized pneumothorax. Bearing these points in mind, we may differentiate the two conditions in most doubtful cases. In some, as we have shown, this is impossible.

[1] Arch. Int. Med., 1917, **20**, 739.

Prognosis.—On the whole, the prognosis in spontaneous pneumothorax during the course of pulmonary tuberculosis is decidedly gloomy, Occurring, as it does, in patients who are already doomed because of the condition of the lungs, this accident but accelerates the inevitable. In very acute cases the patients succumb within a few days, and 90 per cent die within a month. An open pneumothorax, permitting the entry of the contents of pulmonary cavities into the pleura, is almost invariably fatal, sooner or later.

The writer has seen cases of hydro- and pyopneumothorax that have survived for years, and some in whom the fluid has been absorbed, but they are exceedingly rare. Most patients with pyopneumothorax that I have seen have succumbed within one year after the onset of this complication.

Conditions are somewhat different in cases with closed pneumothorax, and also in the localized forms of this condition. They usually occur in patients with slight lesions and with good resisting power. So long as there is no communication with a tuberculous cavity, and the pleura is not infected, as is the case with artificial pneumothorax, the air in the pleura may in time be absorbed. In fact, it was these rare cases of collapse of the lung, and the resulting amelioration of the symptoms of phthisis, which suggested the idea of therapeutic pneumothorax.

CHAPTER XXVIII.

DIFFERENTIAL DIAGNOSIS OF PULMONARY TUBERCULOSIS.

SPEAKING of the diagnosis of pulmonary tuberculosis, some assume that it is only important to differentiate the disease in its early or incipient stage, while when the lesion is more or less advanced the nature of the ailment is so clear that anybody, even of the laity, may make a diagnosis. That this is not the fact is clear when we contemplate the relatively large proportion of non-tuberculous cases admitted to and at times kept for long periods in hospitals for advanced consumptives. Thus, J. Earle Ash[1] found that among the 198 autopsies that have been performed at the Boston Consumptives' Hospital since its foundation, 23 cases, or 11.5 per cent, proved to be non-tuberculous in so far, at least, that no active tuberculous lesion could be discovered. That this is not a unique condition is shown by other figures reported by Ash. He inquired in other hospitals for advanced tuberculous patients in this country and obtained facts about 353 autopsies, among which 38, or 10.8 per cent, were found non-tuberculous. McCrae and Funk[2] have had similar experience: Among 1200 consecutive admissions, all patients coming with a diagnosis of advanced pulmonary tuberculosis, 72, or 6 per cent were found to be non-tuberculous. Of 134 necropsies, 5.2 per cent were found non-tuberculous. Into my service at the Montefiore Hospital there are very frequently sent patients who had spent many months, or even years, in various sanatoriums and hospitals for consumptives, but a careful clinical study of their cases shows that they present no signs of active tuberculosis in any stage or form, and other diseases are diagnosticated, at times confirmed by autopsy.

The number of incipient cases of tuberculosis which, on careful study, prove to be non-tuberculous, is undoubtedly higher. The fact that sanatoriums have a large proportion of "sputum negative" patients, some as high as 50 per cent, testifies strongly in favor of this view. When we bear in mind that hardly more than 10 per cent of "sputum negative" cases—in which the sputum was examined several times and revealed no tubercle bacilli—are actually tuberculous, it is clear that many other clinical conditions pass for tuberculosis very frequently. This was clearly demonstrated in the European armies, in which, at the beginning of the struggle, tens of thousands

[1] Jour. Am. Med. Assn., 1915, **64**, 11.
[2] Ibid., 1919, **72**, 161.

had been rejected by the draft officers and from army hospitals, but a careful examination showed that hardly one-third of these were really tuberculous. To be more exact, of 1000 men suspected of being tuberculous in the French Army, only 1.5 per cent proved to be actually tuberculous, according to Kindberg and Delherm.[1] Eduard Rist[2] reports that in 1000 men sent back to a base hospital as suffering from pulmonary tuberculosis, only in 193 was the diagnosis confirmed by a careful study of the cases. In the rest many other diseases of the lungs, bronchi, and especially the rhinopharynx, were found. In Germany, according to Gerhardt,[3] incipient tuberculosis is diagnosed far more frequently than facts would warrant, both during peace and during war times. Many patients who spent years in sanatoriums were found at autopsy years later with absolutely intact lungs despite the error committed in finding apical lesions while admitting and keeping them in the institutions. A. Fraenkel[4] found that in Heidelberg only 40 per cent, in Halle, only 26 per cent (Hesse), and in Jena as few as 18 per cent were really tuberculous according to Friesicke.[5]

The sufferings inflicted on the patients, their relatives and friends by a diagnosis of tuberculosis, and the stigma it imposes on them, perhaps for life, as well as the economic loss sustained by the individual patient and the community, by such a diagnosis should make us hesitate before pronouncing a case tuberculous. But this can only be done when we have a clear appreciation of the pathological conditions which are likely to be mistaken for tuberculosis. In the following pages will be enumerated and discussed those disease conditions which, in the experience of the writer, are most commonly mistaken for phthisis.

Diseases of the Upper Respiratory Passages.—In the author's experience, the most common pathological conditions mistaken for tuberculosis are diseases of the upper respiratory passages. A large proportion of the "suspects," as well as of the "incipient cases with negative sputum," treated in tuberculosis clinics, and often admitted to sanatoriums where they may be kept for an indefinite time, have no discoverable lesions of any kind in the lungs, bronchi, or trachea. Their main troubles are located in some part of the throat, the tonsils, the pharynx, or one of the nasal sinuses. Many have been operated upon for these conditions one or more times. These patients often cough, expectorate mucopurulent material, at times streaked with blood, etc. During some intercurrent affection, or a subacute exacerbation of the rhinopharyngeal trouble, they may have some fever, anorexia, lose in weight and strength, etc. Streaky sputum at this time is sufficient incentive for a thorough examination. If some

[1] Presse Méd., 1917, **25**, 645.
[2] Jour. Am. Med. Assn., 1917, **69**, 1266.
[3] München. med. Wchnschr., 1918, **66**, 556.
[4] Ibid., 1916, **64**, 1109.
[5] Ibid., 1917, **65**, 1502.

impaired resonance is found in one of the apices—and the right apex is very frequently deficient in air content in these cases—a diagnosis of tuberculosis is made, or at least the patient is placed in the category of the "suspects." Fastidious physicians find in these cases not only impairment of resonance in one of the apices, but also some clicks, or rales provoked by cough, and, perhaps, some prolongation of the expiratory murmur, or even bronchovesicular breath sounds. However, repeated examinations of the sputum fail to reveal the presence of tubercle bacilli. But this does not deter some physicians from making it a case of tuberculosis with negative sputum; some examining physicians for sanatoriums pronounce them tuberculous and admit them to institutions.

In children these chronic rhinopharyngeal conditions, especially adenoids and enlarged tonsils, are even more often responsible for the erroneous diagnosis of tuberculosis, or of tracheobronchial adenitis, because they do not thrive, have mild fever, sweat at the least exertion, or at night when retiring to bed, etc. In fact, in many of these children impairment of resonance may be found in one of the interscapular spaces.

The differential diagnosis of these nasal conditions from tuberculosis is based on one principle which is very important to bear in mind. Tuberculous disease, when active, is accompanied by symptoms of toxemia, particularly fever and tachycardia. At least the temperature and pulse are unstable (see pp. 217, 278). While a slight rise in the temperature may, at times, be discovered in patients with adenoids, hypertrophied tonsils, etc. (see p. 458), it is very uncommon. But the pulse-rate is hardly ever affected in these cases. The cough in rhinopharyngeal disease differs markedly from that of incipient phthisis in most cases. The phthisical subject states that he had never coughed until the onset of the disease, while the patient with rhinopharyngitis has coughed for years, rather mildly hawking up every morning some tenacious sputum, at times streaked with blood, especially during an acute exacerbation; he has been "subject to colds." An examination of the nose and throat usually reveals the source of the trouble—enlarged tonsils, adenoid vegetations in the pharynx, hypertrophied turbinates, chronic sinusitis, atrophic rhinitis, etc. There may also be found some varicosities on the posterior wall of the pharynx, tongue, or trachea (see p. 244) which are the source of the blood in the sputum.

The mistake of pronouncing these patients tuberculous may be avoided in the vast majority of cases by adhering to the following guiding diagnostic principles: *No patient should be pronounced sick with active phthisis unless there are found distinct signs of an apical lesion, with positive sputum, when the pulse and temperature are normal, when he states that he has been "subject to colds" for many years, and shows signs of pathological changes in the nose or throat.* Only constitutional symptoms of phthisis, such as fever, tachycardia, languor, loss of flesh, etc., and tubercle bacilli in the sputum, justify a diagnosis

of tuberculosis when the physical signs of a lesion in the lung are lacking or are indefinite. It is, at times, advisable to send these patients to the country for a few weeks' vacation and it will be found that they improve very rapidly, cease coughing, and gain in weight and strength.

Children with enlarged tonsils or adenoids often show marked rises in temperature every afternoon. Some of the temperature curves of these little patients are not unlike those obtained in tuberculous cases. But we must bear in mind that while subacute and chronic disturbances in the throat are common in children, pulmonary tuberculosis is rare. In addition, children with enlarged tonsils and adenoids, with the lymphatic constitution, are peculiarly resistant to tuberculosis (see p. 588). Moreover, their temperature is unstable, and liable to fluctuations not observed in adults (see p. 458). It is therefore imperative that these factors should be taken into consideration before pronouncing a child tuberculous and perhaps rob it of an education. In children of school age tuberculosis should be diagnosed only when there are definite and clear-cut signs of a lung lesion, especially when the symptoms may be explained as due to evident pathological changes in the nose and throat. Lymphatism in children excludes tuberculosis in an active stage, as a rule. Tuberculous tracheobronchial adenitis, on the other hand, is quite common among these children, but the prognosis is much better than is generally appreciated (see p. 471).

Collapse Induration of the Apex.—In many persons who have been troubled with nasal obstruction for years, certain changes occur, which give physical signs often closely simulating those of tuberculous lesions in the apex of the lung, especially the right.

The symmetry of the two apices is not always perfect, nor do they always have the same resonance and breath sounds in most of apparently healthy people. In many the differences are so striking as to attract attention, and when cough, expectoration, fever, etc., occur for any reason, a diagnosis of tuberculosis is apt to be made based upon the asymmetrical findings over the upper parts of the chest. In persons suffering from adenoids, enlarged tonsils, or other nasal or pharyngeal obstruction, collapse of the parenchyma of the right apex is often met with; the air within the alveoli is resorbed, and the lung tissue becomes indurated, greatly simulating conditions in phthisical lesions. Krönig[1] was the first to describe these cases in detail, and after him many other writers have reported that it is one of the most common respiratory disorders mistaken for phthisis. Many of the negative sputum cases in sanatoriums belong to this class.

It is a purely local, non-specific induration of the lung apex showing physical signs exquisitely simulating those of phthisis. The following points of differentiation may be of value: Patients with collapse induration have suffered from nasal obstruction since childhood, and generally have chronic pharyngitis, enlarged turbinated bones, ade-

[1] Deutsche Klinik, 1907, **11**, 634.

noids, or hypertrophied tonsils. They complain that they have not been able to breathe properly through the nose for years, have expectorated considerably, suffered from dryness and itching of the throat, and have had a strong tendency to colds, tonsillitis, and frequent bronchial catarrh. The classical facies of the mouth breather is often observed in these patients—open mouth, enlarged and drooping lips, obliteration of the nasolabial fold, etc. In tuberculosis all these are lacking. In fact a tuberculous patient with pronounced stigmata of the lymphatic diathesis is exceedingly rare. The sputum shows distinct evidences that it is derived from the upper respiratory tract. It is watery, mixed with saliva, and colorless; sometimes containing gray or bluish globules, not unlike the kind seen in pneumokoniosis. Microscopically there are often found epithelial cells from the mouth, nose and throat, but no tubercle bacilli nor elastic tissue.

Again it must be emphasized that the toxemia of tuberculosis is not observed in these cases. The pulse and temperature are normal, and the nutrition of the patient remains good, excepting during an acute exacerbation of the rhinopharyngeal conditions. The general appearance of the patient is good. Despite the fact that he has been coughing for many months or years, he appears well nourished and does not lose in weight, as is usual in active tuberculosis. He is able to keep at his work efficiently, and the sense of fatigue and languor characteristic of phthisis is lacking.

Apical Catarrh.—Most of us have been warned against the term apical catarrh of a non-tuberculous nature as something which does not exist and should be banished from medical terminology. But it appears that during recent years the profession is again acknowledging that there is often to be seen a catarrhal condition of one or both apices which is not caused by tubercle bacilli. This is especially to be observed in persons who have symptoms and signs of pulmonary emphysema, and those working at dusty trades. They often show all kinds of rales when their apices are auscultated, particularly after vigorous cough due to local bronchitis or tracheitis and to atelectasis. There may be some hoarseness during the morning hours, due to the accumulation of secretions upon the vocal cords, which disappears during the day. The difficulty of differentiating these cases from tuberculosis, especially fibroid phthisis, are often immense. In my wards at the Montefiore Hospital we must, at times, keep these patients for weeks, and examine the sputum many times before we can make up our minds as to the true nature of the trouble. In nearly all cases there is to be observed impaired resonance over one or both apices, but it is to be distinguished from dulness due to tuberculosis by the fact that there is no apical retraction—the resonance above the clavicle usually remains clear, while below the clavicle dulness is elicited as far as the second or third interspace. This is a sign which should always be looked for. Many of these patients are really sufferers from the emphysematous form of fibroid phthisis. But at this stage no special and costly treatment is indicated. It occurs most commonly in persons over forty.

The symptoms of tuberculous toxemia are also lacking; there is no elevation of the temperature nor acceleration of the pulse, excepting in the later stages of the pulmonary emphysema, when there are signs of dilatation of the right heart. The blood-pressure is often high, while in phthisis it is low.

In younger individuals apical catarrh often remains after epidemic influenza. Here the onset suggests an acute exacerbation of a tuberculous process, and the physical signs, combined with the cardinal symptoms, cough, debility, anemia, etc., are very often misleading. But no tubercle bacilli can be discovered, while the constitutional symptoms, fever, tachycardia, etc., are lacking; in fact, after an attack of true influenza the pulse is, as a rule, slow. The prompt recovery of the general health within a few weeks proves that the catarrh is of non-tuberculous origin. Here again we may get a clue by noting that there are no signs of apical retraction, the resonance above the clavicle is normal, and Krönig's field is not contracted in catarrh, while in tuberculosis it usually is. In some cases prolonged observation is required before a positive diagnosis can be made.

Chronic Pneumonic Processes.—Cases which simulate tuberculosis to a degree as to prove baffling are those caused by pulmonary infection with various cocci. In some only observation for many weeks will clear up the diagnosis. The first to make a careful study of these pulmonary infections was Finkler,[1] who found that they are mainly due to various types of streptococci. Recently many others have published extensive clinical and bacteriological studies and have shed considerable light on some of the obscure phases of this condition. From the studied of David Riesman,[2] William Charles White,[3] Louis Hamman and S. Wolman,[4] A. H. Garvin,[5] J. L. Miller, Lorrain, Hans and Gillet,[6] and many others, it appears that we do not deal here with a single distinct pathological process, but that many varieties of infections of the lungs, bronchi, and pleura are classified under this term; the only thing they have in common is that they very frequently simulate pulmonary tuberculosis and are treated as such.

From the clinical standpoint there are two groups to be distinguished —the acute and the chronic types. The writer, at times, has had great difficulty in recognizing those running an acute course, while those of the chronic type, if seen some time after the onset of the disease, are very easily differentiated from pulmonary tuberculosis, usually during a single examination.

The acute cases give a history of a sudden onset with fever, malaise, cough, and pain in some part of the chest. Those that follow epidemic influenza have expectorated more or less blood. Examination at that

[1] Infektion der Lungen durch Streptokokken und Influenzabazillen, Bonn, 1895.
[2] Am. Jour. Med. Sci., 1913, **146**, 313.
[3] Tr. Nat. Assn. for Study and Prev. Tuberc., 1915, **11**, 140.
[4] Ibid., 1916, **12**, 170.
[5] Am. Review of Tuberc., 1917, **1**, 1.
[6] Rev. de méd., 1919, **36**, 173.

time shows no changes in the motion nor the resonance of the chest, but on auscultation feeble, rarely bronchovesicular, breath sounds and moist rales are audible over the greater part of one lobe, usually the lower lobe of the left lung. The apex is only rarely affected, and when this is the case the diagnosis is extremely difficult. Those who believe that in many tuberculous cases the lower lobes are affected at the onset of the disease will at once diagnosticate phthisis when finding signs of a localized lesion in one of the lower lobes. But although there is more or less profuse expectoration of mucopurulent material, no tubercle bacilli can be discovered. On the other hand, all kinds of streptococci and diplococci may be easily demonstrated in every case. The fever abates within a mnth or two, but the physical signs, as well as the cough and expectoration, persist for three or four months, and finally even these disappear, leaving the patient in excellent physical condition.

The differentiation of these cases from phthisis is made first by taking cognizance of the location of the lesion: Basal lesions in tuberculous patients are extremely rare; when they do occur they are terminal phenomena, when the diagnosis is beyond question. *A lesion at the base, while the apex is free, should be considered non-tuberculous unless the sputum is positive as regards tubercle bacilli.* Adhering to this diagnostic principle we may avoid nearly all chances of a mistake of this kind. In the rare cases showing involvement of the upper lobe, it will be noted that the auscultatory phenomena are pronounced, weak, or bronchovesicular breath sounds and showers of rales, localized and persistent in one apex, but percussion yields a resonant, often a slightly tympanitic note. In phthisis such discordance between the findings on auscultation and percussion is extremely rare. When such a large area of the upper lobe is implicated by a tuberculous process there is almost invariably dulness to be elicited above and below the clavicle. Similarly the roentgen-ray findings are, as a rule, negative in non-tuberculous infections of the lung.

The chronic cases usually give a history of an acute or subacute onset. Many of them are, in fact, recurrences of the original acute process. The patient coughs, expectorates more or less sputum, has pain in the chest, and physical examination shows a distinctly localized lesion in one of the lower lobes of the lung, more commonly the left. However, in addition to the fact that the lesion is at the base, thus showing that it is unlikely to be tuberculous, there are other clinical features which tend to show that we are not dealing with tuberculosis. Tuberculosis with such extensive involvement is invariably accompanied by symptoms of toxemia, fever, nightsweats, emaciation, etc., while in the non-tuberculous cases all this is lacking. The temperature is normal, or only slightly elevated some days, and the nutrition of the patient is good. He may be gaining in weight despite the persistence of signs of an extensive lung lesion. The pulse is normal and stable, a point which should always be looked for in these cases.

The low blood-pressure characteristic of phthisis is lacking. Some of these patients, despite the evidence of moisture within the lung, expectorate very little, while others expectorate considerably. Percussion has been of assistance in some cases. When there is no thickened pleura, and this is the case in the majority, the note elicited is resonant, and the *x*-ray findings may also prove negative, while in phthisis with such extensive involvement the reverse is invariably true.

Repeated examinations of the sputum give negative results as regards tubercle bacilli, but pneumococci, or any of the various strains of streptococci, are to be found in nearly every case.

These cases are seen in patients of all age periods. They are very frequent in children of school age, and because of the erroneous statement in many books that the lesion in tuberculous children is most commonly found in the lower lobe, and that negative sputum is the rule, many physicians do not hesitate to diagnosticate tuberculosis in these little patients. But it is worth repeating that *chronic tuberculosis of the lungs in children of school age, when it does occur, affects the upper lobe almost invariably, and lesions in the lower lobes should not be considered tuberculous unless there are symptoms of toxemia and positive sputum.*

The course of these non-tuberculous lung infections is variable. Some recover within a few months and no trace of the trouble can be found. In others, the acute or subacute symptoms recur at irregular intervals, especially after acute "colds" or "grippe" during the winter and autumn months. A large number find their way into sanatoriums, where they remain for months. I have many patients of this class who have taken several "cures" in institutions, and they still show signs of an old lung lesion at one base; they still cough and expectorate, though their general health has been, and is, excellent. Although they have been told that they are sputum-negative cases, a fact which they hardly ever fail to mention, many will not be convinced that their trouble is not of a tuberculous nature.

Chronic Bronchitis and Bronchiectasis.—The average case of chronic bronchitis is easily differentiated from pulmonary tuberculosis when the following points are borne in mind: Barring those in whom the disease is secondary to cardiac or renal disease, and the diagnosis of the primary pathological process is clear, those suffering from bronchitis have been subject to colds, have coughed and expectorated for many years, perhaps since childhood, and their general health has suffered but little, or not at all, as a result of these symptoms. On the other hand, phthisical subjects give a definite history of an onset, be it ever so insidious, when they began to cough, have fever, languor, nightsweats, anorexia, emaciation, etc., symptoms which lack in chronic bronchitis. *We should not be rash in making a diagnosis of tuberculosis in a person who has coughed for many years and his general health has not suffered much, unless the signs and symptoms are clear-cut,*

or the sputum is positive. In chronic bronchitis the harsh bronchial or bronchovesicular breath sounds, if present, are found diffused all over the chest, while in tuberculosis they are localized in only one or in both apices. Similarly adventitious sounds, especially moist rales, in phthisis are found in the upper lobes, while in bronchitis they, are audible over the bases, on both sides of the chest. Despite the fact that the physical signs denote extensive involvement, the general condition of the patient leaves little to be desired, which is never observed in phthisis implicating the lower lobes of the lungs. Tuberculous with basal lesions are hectic, marasmic, and soon moribund; they usually have laryngeal and intestinal complications. In tuberculosis with such profuse expectoration tubercle bacilli and elastic tissue are almost invariably found in the sputum, while in bronchitis repeated examinations prove negative.

Many patients who have been attacked by epidemic influenza with complicating pneumonia remain with chronic or subacute bronchitis for months, and because of the tradition that influenza is an activator of tuberculosis, they are treated as phthisical. Since the recent epidemics many such cases have come under the writer's care. It is, however, to be noted that impairment of resonance, the numerous large, moist, consonating rales are audible, as a rule, over the bases; that the pulse is normal, or rather slow; that the general health is good; in fact, the patients may be gaining in weight despite the extensive pulmonary lesion. The sputum is negative as regards tubercle bacilli, which hardly ever occurs in tuberculous subjects expectorating such large quantities. When these points are borne in mind errors can be avoided in nearly all cases.

Bronchiectasis.—Bronchiectasis is not so easily differentiated, and many patients with this disease pass through life considered tuberculous. Here we have a localized lesion in the chest exquisitely simulating chronic tuberculosis. In fact, many find their way into sanatoriums or hospitals for advanced consumptives, where they are kept for months and years. I have known numerous bronchiectatics who have been admitted to several sanatoriums as far advanced cases with negative sputum. In New York City many are kept under supervision by the authorities, and followed up to their places of employment with a view of preventing the spread of tuberculosis. Among 29,700 patients admitted to the Brompton Hospital, London, during the last twenty years a clinical diagnosis of bronchiectasis was made in 567, or 1.9 per cent, according to Jex-Blake[1]. This author is of the opinion, however, that bronchiectasis was present in fully 5 per cent of the patients admitted.

Bronchiectatic patients give a history of long duration; they have coughed and expectorated for many years, perhaps since an attack of pneumonia or pleurisy; others, since a surgical operation, during

[1] British Med. Jour., 1920, **1**, 591.

which general anesthesia was administered, and they began to cough soon after regaining consciousness. They state that while they almost always expectorate, the cough tortures them only periodically, with frequent remissions of shorter or longer duration, during which they do not cough much, but still expectorate "mouthfuls" without any effort. Posture, as a rule, has an influence on their cough and expectoration; some cough and expectorate more when lying on the right side, while others do so when reclining on the left side. In some the sputum is fetid, while in most it differs but little from that brought out by advanced tuberculous patients. While the majority of bronchiectatics are afebrile, in many careful thermometry reveals low fever, 99.5° F. to 101° F. in the afternoon. But the subnormal temperature in the morning characteristic of phthisis is usually absent in these cases. The pulse is normal, excepting during the advanced stages, when there is cardiac dilatation with tachycardia, dyspnea, cyanosis, etc. The blood-pressure is normal or high, and only rarely low, as is the case in phthisis. A large proportion of these patients spit blood; I have seen many in whom the pulmonary hemorrhage was copious, and even fatal. A blood count will show an increased leukocytosis with a high proportion of polynuclears in bronchectasis, which is rare in phthisis.

It is thus seen that bronchiectasis may easily be mistaken for tuberculosis. The differentiation is made along the following lines: In phthisis tubercle bacilli and elastic tissue are almost invariably found when the sputum is so profuse. Patients with such large, active tuberculous cavities are febrile, cachectic, etc., and often laryngeal and intestinal complications are seen; these are all lacking in bronchiectasis. The tuberculous patient with excavations gives a history of an onset, insidious or acute, with fever, nightsweats, etc., while the bronchiectatic has recently passed through an attack of pleurisy, pneumonia, influenza, etc., or coughed for many years, during which he has not shown any decided symptoms of tuberculous toxemia, and has retained a good general condition of health; he may even be quite adipose despite cough and expectoration. The physical examination, in the vast majority of cases, decides the diagnosis when we bear in mind the following points:

In phthisis the lesion is nearly always localized in the apex or the upper lobe, in bronchiectasis in the lower or middle lobe, most commonly in the left side of the chest, and exceptionally in the upper lobe. In phthisis signs of pulmonary retraction are almost invariably found —deep excavation of the supra- and infraclavicular fossæ; in bronchiectasis these are often fuller than normal. The atrophy of the respiratory muscles characteristic of chronic phthisis is lacking in bronchiectasis. Dulness, when at all discovered by percussion, is found over the upper lobe in phthisis and over the lower lobe posteriorly in bronchiectasis. In the latter, more often than in phthisis, the resonance will be influenced by the presence or absence of secretions in the

cavities—one day when they are full of secretions the note will be dull, while another, after the patient has expectorated profusely, it may be resonant, despite the fact that there are numerous large moist rales. *In the uncommon cases of bronchiectasis of the upper lobe there is elicited resonance above the clavicle and dulness below it; the reverse is almost invariable in phthisis.* With large tuberculous cavities in one lung there are almost always signs of implication of the other side of the chest, while bronchiectasis is commonly unilateral. If the case has been kept under observation for a long time it will be noted that in tuberculosis signs of consolidation precede those of excavation, while in bronchiectasis the reverse generally occurs. Bronchiectatic cavities remain of about the same size for many years, often indefinitely, while active tuberculous cavities, with large, moist rales, show a decided tendency to enlarge. When bronchiectatic cavities involve both sides of the chest, which is very rare, they are found irregularly scattered, with almost normal lung tissue intervening, while phthisical cavities are contiguous extensions of the original apical lesion. It is, in fact, very rare to find in phthisical chests signs of more than one cavity with more or less healthy lung tissue between the two excavations. In multiple bronchiectasis this is the rule. The heart is found displaced in both phthisis and bronchiectasis toward the affected side, but there is one significant difference which has been pointed out by William Ewart: "The displacement of the heart toward the diseased side in the chest in the usual cases of unilateral phthisis follows an oblique direction upward; a horizontal displacement is exceptional and suggests some complicating pleural factor. In unilateral bronchiectasis the displacement is, practically speaking, always horizontal; not only by reason of the basic origin of the disease, but largely also owing to the lowering of the diaphragm on the sound side, with extension of the cardiac beat into the epigastric notch." This holds true in the vast majority of cases, and is most easily determined with the aid of the roentgen-rays.

Another important distinction is to be mentioned: In bronchiectasis the breath sounds are usually feeble, though in some instances they may be bronchial; a large number of coarse, moist, consonating rales are audible, and the *x*-rays show only slight opacities, variable, according to the fulness or emptiness of the cavities at the time the examination is made. In phthisical cavities the *x*-rays findings are more accentuated and more extensive than physical signs would lead us to suppose. Scoliosis is a common sign of bronchiectasis—the convexity is turned toward the affected side; this is rare in cases with phthisical cavities.

Pleural Vomicæ.—Among the non-tuberculous pulmonary conditions treated as advanced consumption, and often sent in to my wards at the Montefiore Hospital, are pleural vomicæ—localized collections of pus in the pleural cavity, originating in the lung, pleura, or even the abdominal organs, but burrowing their way to the exterior through a bronchus or fistula. These vomicæ may be found anywhere in the

chest, but they are most commonly located in the region of the interlobar fissure, and at the diaphragmatic pleura.

The differential diagnosis between pleural vomicæ and advanced phthisis is very simple in the vast majority of cases. Of first importance is a good history of the case. Pleural vomicæ begin acutely with symptoms of pleurisy with effusion. After a variable time the acute symptoms abate, and the patient recovers to a certain degree, but he keeps on expectorating large quantities, "mouthfuls," of purulent matter. In others, there is a history of pneumonia, followed by empyema which broke through a bronchus. In still others, there is a history of an acute septic process, especially after a surgical operation, or after childbirth. Cases have been observed in which the pus in the pleural pocket came from an appendicular or hepatic abscess, the pus burrowing its way into the pleura, and then through the lung into a bronchus to the exterior.

All cases give a history of an acute disease with fever, perhaps of the septic type, prostration, pain in the chest, and either primary or secondary pneumonia or pleurisy was diagnosticated at the time. The fever lasted a variable time, in some cases several weeks, when it suddenly dropped with the appearance of profuse expectoration of purulent material. The expectoration may come on suddenly with a gush, almost asphyxiating the patient. During the first few days the amount brought out is considerable. In a case under my care it was more than a pint. Within a few days the amount of sputum decreases, but it still remains relatively profuse for an indefinite time. It is during this chronic stage that tuberculosis is often diagnosticated. These patients cough, expectorate purulent sputum, often have hemoptysis, are emaciated, and run a subfebrile temperature, influenced by the amount of expectoration. When the latter is profuse, the fever is negligible, but during days when the communicating bronchus is plugged, the fever is high. The cough and expectoration are influenced by posture; some cough more when lying on the affected side, while with others the reverse is true, apparently depending on the direction of the communicating fistula or bronchus.

Physical exploration of the chest shows signs of an extensive basal lesion, usually simulating the signs of a pleural effusion. In fact the first thing one thinks of after going over such a chest is fluid, probably pus. But exploratory puncture fails to confirm it. In the cases in which the vomica is located in the region of the interlobar fissure, the signs are those of consolidation, or excavation of the upper part of the lung, though careful examination shows that the real apex of the lung remains unaffected, a fact which is of great diagnostic significance. The affected area is dull, more commonly flat, on percussion, and either feeble, cavernous, or amphoric breath sounds are heard, depending on whether the cavity is filled with secretions or empty at the time of the examination. Large, moist rales, of a consonating character, are audible over the affected area.

The differential diagnosis between this condition and phthisis is thus clear: The history points to an acute onset, as pleurisy or pneumonia, or following some other septic process; the lesion is localized in the lower lobe, or in the region of the interlobar septum, while the apex of the lung remains free. These, combined with the fact that, despite its abundance, the sputum is negative, rules out tuberculosis. We have repeatedly emphasized that *when the apex shows no signs of a lesion only positive sputum should justify the diagnosis of phthisis.*

Abscess of the Lung.—At times patients with pulmonary abscess, especially the chronic form, are treated as advanced consumptives. A consideration of the history of the case should clear up the diagnosis in most cases. It is often preceded by an attack of aspiration pneumonia, mainly after an operation, very often after tonsillectomy, or after a septic pulmonary embolism. There are hectic fever, sweating, emaciation, and spasmodic cough, expelling large quantities of sputum which differs, as a rule, from that observed in phthisis. It has a brown color, due to an abundance of hematoidin crystals and elastic tissue fibers which may be found microscopically. In rare instances it is fetid, but not as offensive as that of gangrene of the lung. Hemoptysis, at times copious hemorrhages, occur, and it is often this symptom that suggests phthisis to the patient and the physician. But here again the location of the lesion should clear up the diagnosis. It is usually in the lower lobe, while the apex remains free from pronounced pathological changes. I have seen several cases in which the lesion was in the upper lobe, especially in diabetics, or in persons on whom operations on the lower jaw were performed. Many of my cases began soon after operations on the tonsils. But all in all, the history, the fact that the apex is free, the character of the sputum, and the absence of tubercle bacilli, are diagnostic points.

Gangrene of the Lung.—When occurring as a sequel to aspiration pneumonia, pulmonary embolism, or after operations on the jaw in diabetics, or to the entry into the bronchi of foreign bodies such as teeth, fishbones, etc., gangrene of the lung is easily distinguished from phthisis by the history alone. But when occurring as a complication of bronchiectasis it is, at times, mistaken for tuberculosis: The history of cough and expectoration for many years is apt to prove misleading. When severe pulmonary hemorrhage is one of the symptoms, the diagnosis of tuberculosis is fortified.

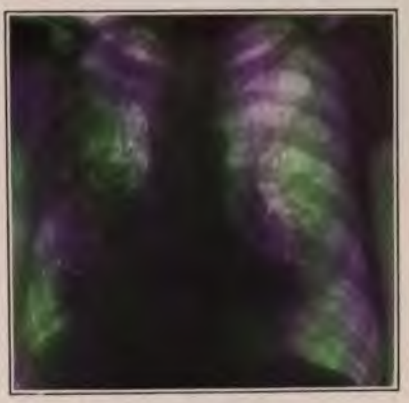
But fetid sputum is exceedingly rare in phthisis; as rare as gangrene is as a complication of phthisis (see p. 563). Gangrene is characterized by high irregular fever, prostration, cough, and expectoration of considerable quantities of fluid, frothy, and highly offensive sputum which separates into three layers, the lowermost containing fragments of lung tissue. Elastic tissue is only rarely found in the sputum because it is soon destroyed by the rapid action of the pathogenic agent, but at times fragments of lung tissue may be discovered. None of these clinical features are seen in pulmonary tuberculosis, excepting when gangrene appears as a complication.

PLATE XXIV

FIG. 1

Pleural vomica. Dense homogeneous shadow in lower third of left side of the chest. Several dilated bronchi in left hilus region. Obliteration of left costophrenic sinus and displacement of the heart to the left. Note the absence of changes in the apices.

FIG. 2



Multiple bronchiectasis. Diffuse shadow in right hilus, middle lobe and portions of axillary regions, studded with bronchiectatic cavities. Obliteration of costophrenic sinus; pleuropericardial adhesions. Marked peribronchial changes in left lung. Supraclavicular fields practically free from changes.

FIG. 3

Bronchiectasis. Large cavity with thick walls in upper lobe of right lung; thick pleura with dilated bronchi in lower lobe; elevation of diaphragm. Left lung emphysematous; calcified nodule in second interspace. Dextrocardia.

FIG. 4

Metastatic hypernephroma of the lung. Autopsy. Sent in as tuberculous. Opacities denoting infiltration of both apices. Effusion (hemorrhagic) into right pleura.

PLATE XXV

Fig. 1

Fig. 2

Fig. 3

Fig. 4

Malignant tumor of the left lung. In the first roentgenogram the shadow could hardly be differentiated from a tuberculous lesion. It was only in the third roentgenogram, taken three months later, that the true nature of the affection could be made out roentgenographically.

Cancer of the Lung.—Intrathoracic neoplasms, especially carcinoma and sarcoma of the bronchi, lung, pleura, and glands, are often mistaken for tuberculous disease of the lung. The onset is insidious with cough and mild fever, the curve in some cases under my care having exquisitely simulated that seen in typical cases of incipient tuberculosis. When to this are added hemoptysis of various degrees, and loss in weight, it is clear that there are strong reasons for thinking of tuberculosis, the more common disease. Moreover, malignant disease of the lung is likely to pursue a very slow course.

The differentiation is made by the symptomatology, and the physical signs as well as with the aid of the *x*-rays, though I consider the *x*-ray findings less reliable in early cases than rational interpretation of signs elicited by exploration of the chest (see Plates XXIV and XXV). While growing, the tumor gives rise to certain pressure symptoms which are of immense value in the differential diagnosis. Pressing upon the superior vena cava, enlarged veins on the chest wall and shoulder or anterior part of the neck are produced; pressure on the sympathetic will dilate the pupil on the affected side. There may be a difference in the fulness of the pulse when the two radials are compared. These signs are invaluable, but they are more often absent as present.

At the beginning of the disease, when the new growth is yet insignificantly small, the physical signs may not show any alterations in the resonance and breath sounds, and the constitutional symptoms of tuberculosis may be so striking as to mislead. But it appears that in nearly all cases, even those showing a subfebrile temperature, the pulse is normal, which is rare in tuberculosis. Pain in the chest, and tenderness of the skin of the affected side are characteristic of tumor, but rare in phthisis. Dyspnea is very common in tumor, rare in phthisis. In some cases I have noted symptoms and signs of pulmonary emphysema, with slight fever and hemoptysis. Here again the diagnosis was difficult and required prolonged observation before a conclusion could be arrived at.

With the growth of the tumor, local signs may be made out by physical examination. If the neoplasm is located in the upper part of the chest, the signs again simulate tuberculosis, but a careful analysis of the findings usually shows striking differences. Emaciation appears early in phthisis, while in cancer of the lung the nutrition of the patient may leave little to be desired for many months. Even in the later stages, when the patient loses in weight considerably and progressively, the cachexia differs markedly from that of tuberculosis. In the latter the patient appears hectic, while in the former the waxy yellow tinge of cancerous cachexia is almost invariably noted at first sight; the severe blanching of the face also betrays malignant disease. But, as was already stated, cachexia appears late in intrathoracic tumor, while in tuberculosis it is often an early symptom.

Percussion over the site of the tumor elicits a flat note, which is never found in tuberculosis of the upper lobe. Moreover, above the

area of flatness there may be an area of resonance, again unknown in tuberculous lesions. The affected side of the chest in many cases may thus be found to be made up of three zones: an upper, resonant one, above the second rib; a middle one, from the second rib for one or two interspaces, which is flat, and the lowermost again resonant. The upper area of resonance should immediately excite suspicion. When the growth appears first in the lower parts of the chest tuberculosis should not at all he thought of, but often pleural effusion is simulated, but this is excluded very easily, in most cases by the history, course, and physical signs.

Auscultation also gives very valuable clues. If the tumor is of some dimensions, there will be noted feeble or complete absence of breath sounds over the circumscribed area which has been found flat on percussion. Now, in tuberculous lesions with such extensive implication there are almost invariably to be heard adventitious sounds, usually large, moist, and consonating rales. It may be stated that *a very dull or flat note, with feeble breath sounds, without any rales is strongly suggestive of a tumor*. The reasons for the absence of breath sounds are these: The tumor often arises from the wall of a large bronchus and with its growth it compresses the air tube; or when one of the mediastinal glands is the source of the tumor, its growth may compress a large bronchus in its vicinity. The result is atelectasis of the lobe supplied by this bronchus—flatness, absent breath sounds, etc., but no rales. Again, such extensive tuberculous involvement is accompanied by adventitious sounds. These signs, combined with the lack of the toxic symptoms of phthisis, especially the lack of tachycardia, are sufficient to direct attention to the problem of malignancy, even in the presence of such symptoms as cough, slight fever, hemoptysis, etc.

With the growth of the tumor the area of flatness increases, the veins on the chest become more and more prominent, and at times metastatic deposits are discovered in the glands above the clavicles, etc. A good sign to be considered is the position of the trachea and the heart: In tuberculosis with such extensive involvement these two organs are drawn toward the affected side, while in cancer of the lung they are pushed away toward the unaffected side. This sign has served me often in doubtful cases. In the later stages of cancer, when the tumor become gangrenous, and fetid sputum is expectorated, the mediastinum may be drawn toward the affected side, as has been noted by the writer in rare instances.

Malignant tumors of the pleura or lung are complicated by pleural effusions in over 50 per cent of cases. In many the fluid is serous, in others, serosanguineous, while at times it is purulent. If the diagnosis has not been previously made the difficulties increase when this occurs. The fluid is often sanguineous, but this does not help us, because it is often so in phthisis. The pressure signs enumerated above are, however, more likely to occur in cancer. A careful watch for metastases may clear up an otherwise obscure case. We must, however, again

emphasize that when blood is found at the first exploratory puncture, it is of greater diagnostic significance than when found at the second puncture, because tapping the chest, at times, causes bleeding, thus coloring the effusion. Purulent effusions are not uncommon in cancer. At times fragments of the growth or characteristic cells are found in the centrifuged specimen of the aspirated fluid, but this is rare in my experience. It has been stated that when a large number of coarsely granular eosinophile cells are found in the fluid it is a good sign of tumor of the pleura or lung.

One characteristic of effusions in cases of cancer of the lung is that they fill the entire pleural cavity; at least, flatness is elicited while percussing the chest from above the clavicle down to the diaphragm, and this is a sign which has hardly ever failed the writer in cases in which fluid in the chest was the first thing seen when the patient is examined. In inflammatory pleural effusions the resonance above the upper level of the fluid is more or less tympanitic—skodaic. In hydro- and pyopneumothorax, it is shifting. In cancer of the pleura or lung it apparently fills the entire chest, which is exceedingly rare, if it ever occurs, in effusions due to other causes in which there is at least resonance above the clavicle.

The writer[1] has recently aspirated fluid in the chest and replaced it with air in the usual way in which a pneumothorax for therapeutic purposes is induced. A tumor is then made much clearer on the roentgenographic plate than can be expected otherwise. Similarly a diagnostic pneumothorax is induced in cases without fluid with a view of bringing out the outlines of an intrathoracic neoplasm.

I have observed several in cases in which the first symptoms and signs were those of pulmonary emphysema, only the persistent unproductive cough, slight fever, and hemoptysis drawing the attention of the patient to the seriousness of the disease. The dyspnea may be severe, and in one case it was even distinctly stridorous, due to pressure of the growth or the implicated tracheal glands on the trachea. In a few cases I have seen distinct pulsations of the thorax, especially when the tumor appeared in the upper part of the chest. These pulsations could be perceived by palpation. These signs are not all present in every patient, still some may be found in each case, and should be considered before a diagnosis is made.

Actinomycosis.—Actinomycosis of the lung, in its initial stages, presents symptoms and signs not unlike those of pulmonary tuberculosis. There are cough, loss of flesh, mild fever, etc. So long as the fungus remains within the lung there may be little or no expectoration and the sputum is microscopically negative. When it makes its way into a bronchus, a microscopic examination may reveal the fungus, if it is looked for. When it reaches the pleura, symptoms of pleurisy with effusion arise and the patient is for a time treated for tuberculous

[1] Jour. Am. Med. Assn., 1921, **76**, 581.

pleurisy or empyema. The constitutional symptoms in advanced cases of actinomycosis simulate those of active and advanced phthisis very strikingly. There are hectic fever, tachycardia, emaciation, cough, and expectoration of large quantities of nummular sputum, hemoptysis, etc.

In the initial and latent stages actinomycosis differs from phthisis in the following points: Tuberculosis lesions begin at the apex almost invariably, while actinomycosis is usually localized in the middle or lower lobe. We have already repeatedly warned against a diagnosis of tuberculosis when the apex remains unaffected. There are, however, cases of actinomycosis in which the upper lobe of the lung is the first to be affected. Because at this stage tubercle bacilli and elastic tissue may be absent in phthisical patients, the difficulties are at times unsurmountable. A careful search should be made for the ray fungus in all doubtful cases.

The diagnosis is usually cleared up within a few weeks when a fluctuating swelling appears on the chest wall, which may suggest empyema necessitatis, especially since there are also signs of a pleural effusion. But an examination of the sputum, or of the pus removed from the external swelling, shows yellowish granules or streaks of actinomycotic growth.

Streptotrichosis of the Lung.—Nocardia.—Infection with any of the microörganisms of the streptothrix group may give symptoms which are often mistaken for those of chronic pulmonary tuberculosis. Of late, many cases have been reported in this country, while in Europe these infections have been described as *pseudotuberculosis*. In 1898 Simon Flexner[1] described a case of this "pseudotuberculosis" with autopsy. More recently Edith J. Claypole,[2] William M. Stockwell,[3] and others, have reported cases in this country.

The symptoms are those of chronic phthisis. The onset is slow and insidious. The patient coughs, expectorates mucopurulent material, and is short-winded. Hemoptysis is not uncommon though profuse pulmonary hemorrhages are not observed. Either streaky sputum or small amounts of blood are brought out. The symptoms of toxemia, such as fever, nightsweats, tachycardia, etc., are usually wanting. Most patients present a rather good external appearance despite the symptoms of pulmonary trouble which may have lasted for years. It seems that most patients are treated as tuberculous with negative sputum, and are admitted to sanatoriums and hospitals for advanced consumptives.

The differential diagnosis can only be made by a microscopical examination of the sputum, of which large quantities should be obtained for the purpose. Inasmuch as the staining methods employed to discover tubercle bacilli render the streptothrix invisible, and some of

[1] Jour. Exper. Med., 1898, **3**, 435.
[2] Arch. Int. Med., 1914, **14**, 104.
[3] Tr. Nat. Assn. for Study and Prev. Tubere., 1916, **12**, 265.

the stains are acid-alcohol-fast, great care should be taken in gauging the amount of the decolorization of the carbol-fuchsin preparations, which should be varied so as to differentiate the less acid-fast types. The Gram-method of staining may also be employed. There are strong reasons for believing that if special care were taken with all sputum-negative cases showing signs of chronic tuberculosis, more cases of pulmonary streptotrichosis would be discovered. Claypole[1] believes that she has worked out a certain serological reaction which she recommended as of diagnostic value in streptothrix, but considering the large number of types of this microörganism, it is problematical whether a single skin reaction will be efficient diagnostically.

Bronchopulmonary Spirochetosis.—Hemorrhagic Bronchitis.—This disease was first observed in Ceylon and described by A. Castellani in 1905. Later Jackson reported cases in the Philippine Islands, and during the World War, many cases were discovered among the troops in France, Belgium, Italy, England, Switzerland, etc. In a soldiers' sanatorium in Northern Italy, Castellani[2] found that 3 per cent of the patients sent in with the diagnosis of pulmonary tuberculosis, in reality suffered from bronchopulmonary spirochetosis. Similar experiences have been recorded in France.

The symptoms are akin to those of pulmonary tuberculosis, and most patients are treated as such. As given by H. Violle,[3] there is cough, more or less copious expectoration, but fever is lacking in the majority of cases, while the general condition of the patient leaves little to be desired. Violle states that the gross appearance of the sputum is characteristic and that a diagnosis can be made by examining it alone. The expectoration is viscid, uniformly thick and closely resembles the juice of gooseberries. But what makes the symptomatology like that of phthisis is the pulmonary hemorrhage which is never lacking. The blood brought out may be considerable and is of a peculiar pinkish color; fatal hemorrhage, however, never occurs. P. Nolf and P. Spehl[4] describe cases without hemoptysis. If these cases the sputum was mucopurulent, yellowish-green in color, and after some days became fetid, the fetor remaining *pari passu* with the number of spirilla.

Physical exploration of the chest may reveal nothing, or some signs of bronchitis may be discovered. In rare cases signs of consolidation of an apex or any part of the lung have been found. S. Fishera[5] reports cases of apical catarrh running a chronic course with fever, loss of flesh, nightsweats, and blood-stained sputum containing spirochetæ, but no tubercle bacilli.

Several cases were admitted to the Montefiore Hospital as tuberculous.[6] The symptoms and signs were those of a pulmonary abscess

[1] Jour. Exper. Med., 1913, **17**, 99.
[2] Presse Méd., 1917, **25**, 377.
[3] Bull. Acad. de Méd., Paris, 1918, **79**, 429; Lancet, 1918, **2**, 775.
[4] Arch. Méd. Belges, 1918, **71**, 1.
[5] Riforma Medica, 1918, **34**, 384.
[6] Fishberg and Kline, Arch. Int. Med., 1921, **27**, 61.

with pain in the chest, high continuous fever, incessant cough and copious expectoration of offensively fetid sputum. Spirochetæ and fusiform bacilli were found in the sputum, and, at the autopsy, in the pulmonary tissues. However, these organisms were also found in the gums of patients who suffered from severe pyorrhea, and the impression was gained that the lungs, at least in these cases, were infected by the spirochetæ derived from the buccal mucous membrane. In another case in which these organisms were found in the sputum there was also pyorrhea. This patient suffered from a chronic bronchial disease, and the signs were not unlike those of fibroid phthisis.

The differentiation from phthisis can be made only by a microscopic examination of the sputum. The *spirochetæ bronchialis* and fusiform bacilli are found in large numbers; in some cases the specimens are actually teeming with them. They may be stained with the Romanowski stain, but the silver nitrate stain of Fontana-Tribeudeau is superior. The organism is extremely variable in shape, length, and number of spirals. It has not yet been cultivated, but Chalmers and O'Farrell have succeeded in inoculating monkeys by intratracheal injections of a patient's sputum. The prognosis is generally favorable in acute cases, though in those developing pulmonary abscess, as the one observed by the writer, a fatal issue is the rule, but when the disease runs a chronic course it may last indefinitely, with occasional remissions.

The differentiation from phthisis is thus made by the following criteria: Absence of tubercle bacilli, of hyphomycetes, and of ova of *Paragonimus westermanii* from the sputum, while the *Spirochetæ bronchialis* and fusiform bacilli are found.

Pulmonary Lesions in Cardiac Patients.—Patients suffering from organic heart disease, especially mitral stenosis, often cough, expectorate, spit blood, have mild fever, and are emaciated, and for these reasons are very frequently treated for tuberculosis. Several cases of mitral stenosis are annually sent into my wards in the Montefiore Hospital as tuberculous, and in my private practice I have very frequently cases presented to me as tuberculous, though they only suffer from mitral obstruction. The main reason is the frequency of hemoptysis in mitral stenosis, which is, as has already been mentioned, next in frequency to tuberculosis a cause of blood spitting. The amount of blood expectorated may be slight, only streaky, and in rare cases even copious. I have observed it in fully compensated cardiac lesions, and in those with signs of decompensation. In the former, the hypertrophied heart pumps the blood through the pulmonary vessels with great vigor, and because of the obstruction it meets while passing through the narrowed mitral valve the pressure is increased, rupturing some of the capillaries. It is for this reason that we meet at times with hemoptysis in patients in whom compensation is as perfect as could be expected. In cases of heart failure also hemoptysis occurs, at times, due to hemorrhagic infarction or embolism; though embolism may be said to be an infrequent cause, and when it does occur, it is due to an

antemortem clot in the right auricular appendix. The symptoms are clear-cut, but I have seen many in which tuberculosis was diagnosticated. It usually occurs suddenly, producing acute pain in the chest, dyspnea, orthopnea, cyanosis, and hemorrhage, at times very copious. In other cases the bleeding is due to thrombosis of the pulmonary vessels, and then the accompanying symptoms are less acute.

In rare instances *hoarseness* in addition to the cough and hemoptysis may suggest tuberculosis when the real trouble is mitral stenosis. Osler,[1] Frischauer, Alexander, Hofbauer, and others, have drawn attention to hoarseness, and a brassy or stenotic cough as a symptom of mitral stenosis. In a case reported by Osler it was found at the autopsy that the left recurrent laryngeal nerve was compressed against the arch of the aorta and the ligamentum Botalli by the dilated left auricle and has shown degenerative changes.

In chronic cases of mitral stenosis in which cough, emaciation, hemoptysis, etc., suggest a tuberculous process the following points of differentiation are to be borne in mind: Dyspnea on exertion is more pronounced in cardiac than in pulmonary patients. We have already shown that *dyspnea is not one of the cardinal symptoms of phthisis in its early stage* (see p. 278). Rest for a few days may relieve the dyspnea of cardiacs. The cough in cardiacs is aggravated during the cold weather, or when the patient walks against the wind, and not influenced much by sedative medication (heroin, codein, etc.) which relieve the cough in the tuberculous. Digitalis, however, often relieves the cough in cardiacs. In the tuberculous the heart is smaller, at least not larger, than normal, while hypertrophy or dilatation may be made out in nearly all cases of mitral stenosis.

Physical exploration of the chest may show some areas of atelectasis, catarrh, or localized pulmonary edema, simulating phthisical lesions. Owing to brown induration, the signs elicited over the apical area are those of consolidation, in some cases. In most cases, however, the resonance above the clavicle is not impaired. Rales, when heard, are found over the lower parts of the chest and bilaterally, and they are not constant, because they are due to localized edema of the lung. The cardiac murmur characteristic of mitral stenosis usually decides the diagnosis. In the rare cases of mitral stenosis without murmurs, or when the murmur disappears owing to decompensation, we usually find an accentuation or reduplication of the second sound in the second intercostal space near the left border of the sternum. At the apex the first sound often has a snapping character and on palpation a systolic tap or shock may be felt. Percussion shows enlargement of the area of cardiac dulness, and in cases of decompensation some form of arrhythmia, usually that of auricular fibrillation, may be noted, all of which are lacking in phthisis.

These signs should be sought for in every case of cough and hemop-

[1] Arch. gén. des malad. d. coeur, 1909, **53**, 35; Jour. Am. Med. Assn., 1908, **4**, 205.

tysis in which the signs of pulmonary tuberculosis are not clearly noted. While it is possible that patients with mitral stenosis should become tuberculous, yet this is exceedingly rare. In fact, *it has been my rule never to diagnosticate tuberculosis in one showing signs of disease of the mitral valve and cardiac hypertrophy or dilatation, irrespective of the physical signs elicited while examining the lungs, unless the sputum reveals tubercle bacilli.* I have hardly seen more than a dozen cases of mitral stenosis developing phthisis.

Pulmonary infarction occurring during the course of cardiac disease, or from an embolus arriving from some distant diseased vein, may be a source of error, as I have seen in several cases. The patient knows that he has an organic heart lesion, or phlebitis, and perhaps has been treated for these conditions. Suddenly, without any warning, he is seized with severe pain in the chest, distressing dyspnea, or orthopnea, and hemoptysis. In some the bleeding is very copious, even threatening life. After the acute symptoms have been ameliorated, an examination shows signs of a localized area of diseased lung: Impaired resonance, feeble, or bronchial, breath sounds, and moist rales. These physical signs are mostly found in one of the lower lobes, but may also occur in the middle or upper lobes, especially in the interscapular space. But the history, as well as the signs of a cardiac lesion, or of phlebitis, should clear up the diagnosis in most cases. However, I have seen many patients with mitral stenosis, or with remnants of pulmonary infarction, treated for an indefinite period in tuberculosis clinics in New York City, and others who have been admitted to sanatoriums and kept there for months.

In *acute endocarditis* and *pericarditis*, rheumatic or infectious, symptoms of tuberculosis may be present. There are fever, tachycardia, emaciation, hemoptysis, and some of the physical signs of acute miliary tuberculosis. In most cases no murmur is audible, and the area of cardiac dulness may not be found increased perceptibly. When to all this there is added a pleural effusion, which is not uncommon, the diagnosis of tuberculosis appears inevitable. But a careful inquiry into the history of the onset of the disease, as well as the fact that the pleural effusion is bilateral, should excite suspicion. Patients with acute articular rheumatism, when showing some signs or symptoms of tuberculosis, should not be considered tuberculous without positive proof, or a careful study of the course of the disease. Signs of pericardial effusion are also indications that we are dealing with a cardiac, and not with a tuberculous lesion.

Syphilis of the Lung.—Syphilis of the lung is an extremely rare disease, and when it does occur it is very difficult of diagnosis pathologically as well as clinically. According to Osler,[1] of 2500 autopsies at Johns Hopkins Hospital, lesions which were believed to be syphilitic were present only in 12 cases. In a study which included all the

[1] D'Arcy Powers' System of Syphilis, London, 1914, **3**, 15.

PLATE XXVI

FIG. 1

Syphilis of the lung simulating in the roentgenogram a tuberculous lesion in the right apex.

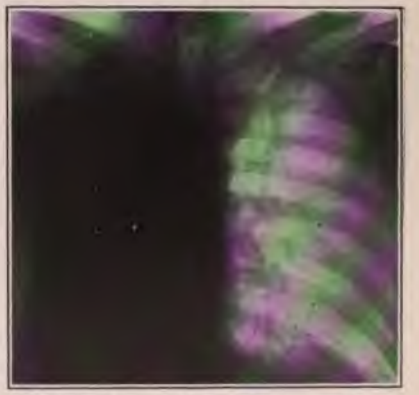
FIG. 2

Pulmonary syphilis. Diffuse peribronchial infiltrations of right lung, mostly marked at the lower half. Hilus glands in left lung are distinctly enlarged. Pericardial adhesions mainly seen in right side.

FIG. 3

Moderate calcification at the hilus on both sides. Right diaphragm elevated. Heart enlarged, aorta dilated. Both apices free. Clinical diagnosis, syphilis of the lung. Admitted as tuberculous, and treated as such for many years.

FIG. 4



Malignant growth; empyema. Homogeneous shadow obscuring left lung field. Because of the fluid and the heart is pulled toward the left, the roentgenogram cannot decide whether it is due to a thick pleura and parenchymatous lesion, or solely to effusion. Right lung emphysematous and also shows a slight infiltration of the apex.

London Museums, J. K. Fowler[1] was only able to discover 12 specimens, and 2 of these were of a doubtful nature. Among 6000 cases of syphilis at the hospital at Copenhagen, syphilis of the lung was observed only in 2; and among 18 patients with acquired syphilis who came to autopsy, gummatous lesions of the lung were found 3 times. Chiari[2] found only 2 cases of syphilitic lesions of the trachea and bronchi, and 1 of syphilis of the lung. Petersen among 88 autopsies of patients with acquired syphilis found lung lesions only in 11. The rarity of pulmonary syphilis, despite the fact that syphilis is so widespread, testifies that errors in diagnosis are at least as rare as the disease. But now and then we meet with a case which shows symptoms simulating pulmonary tuberculosis and treated as such.

Syphilis of the lung manifests itself by the usual symptoms of chronic pulmonary tuberculosis, such as cough, expectoration, slight fever, loss in weight, and at times even hemoptysis. But it appears that in nearly all cases the course of the disease is rather slow; in none of the cases observed by the writer has the disease pursued a progressive course, nor has it perceptibly disabled the patient. Physical exploration of the chest shows that the lesion is localized in the lower or middle lobe, and the apex remains practically free from changes. This alone should excite suspicion that it is not tuberculous. A careful search should be made for the stigmata of syphilis in the bones, skin, larynx, rhinopharynx, eyes, etc. The Wassermann reaction may be of help, but not so much as would be anticipated, because it is frequently positive in tuberculosis, and phthisical subjects may have had syphilis. In fact, the two diseases are found concurrently very frequently. (See p. 598.) Absence of tubercle bacilli from the sputum is no criterion, because in really syphilitic phthisis the amount expectorated is rather scanty, at least in the early stages.

The differentiation is best made by the application of the therapeutic test. Properly administered doses of arsphenamine, mercury, or iodide of potassium will promptly remove the symptoms of syphilitic phthisis. In some of my cases the effect was very prompt, within a couple of weeks the cough disappeared, weight and strength returned, and the patient considered himself well. But this does not imply that in the least suspicious case a diagnosis of syphilis of the lung should be made and treatment applied. Patients with pulmonary tuberculosis are often harmed by antisyphilitic treatment, especially mercury and the iodides. Considering the extreme rarity of syphilis of the lung, it is clear that Fowler's suggestions should always be borne in mind: (1) The cases must be complete, that is, the symptoms observed during life must be considered in connection with the lesions discovered on postmortem examination. (2) The evidence of syphilitic infection must be undoubted. (3) Repeated examinations of the

[1] Diseases of the Lungs, London, 1898.

[2] Quoted from F. Balzer, Brouardel-Gilbert-Thoinot, Traité de Médecine, Paris, 1910, **29**, 641.

sputum must have been made, and tubercle bacilli have been invariably absent, and the absence of tubercle from the lungs as the cause of the lesions must be proved by postmortem examination. (4) Syphilitic lesions about the nature of which there can be no doubt must be found in other organs.

Many tuberculous patients also suffer from syphilis, as has already been mentioned. When tubercle bacilli are implanted on a syphilitic subject the course of phthisis is rather favorably influenced, probably because it is characterized by a tendency to the production of connective tissue (see p. 598). It must always be borne in mind that the presence of syphilis does not exclude phthisis, but that the latter is very often engrafted on the former.

Hyperthyroidism.—The syndrome of hyperthyroidism, which is so commonly met with in young persons, and in women during the menopause, is very frequently mistaken for phthisis. The acceleration of the pulse-rate, the frequent sweating at the least provocation, the slightly elevated and unstable temperature and the tendency to fatigue and languor, emaciation, and in women menstrual disturbances, are suggestive of the symptoms of early phthisis, especially when the patient coughs for any reason. On the other hand, symptoms and signs of disturbance of the autonomic nervous system are very frequently seen in phthisis, as has recently been shown from studies of Meyer Solis-Cohen,[1] Sabourin,[2] Sergent,[3] Boas, and others. Many cases of the milder grades of hyperthyroidism are therefore treated for tuberculosis.

The severe cases of this syndrome, those showing the cardinal signs of Graves's disease, goiter, tachycardia, tremor, and exophthalmus are not likely to be mistaken for phthisis, unless the latter appears as a complication of the former. But mistakes may be avoided even in the milder forms of hyperthyroidism when the following points are considered: Patients with tachycardia, liability to sweat at the least exertion or excitement, languor, dermographism, etc., are not to be considered tuberculous unless a physical examination of the chest, and perhaps roentgenography, reveals a distinct pulmonary lesion. In doubtful cases it may be advisable to wait for the results of repeated sputum examinations. Otherwise the characteristic symptom of hyperthyroidism, rapid heart action, palpitation, fatigue, flushes, sweats more during the day than during the night, a slight tremor of the fingers, etc., are sufficient to define the nature of the trouble. In some cases, showing signs of collapse induration (see p. 537), only prolonged observation will clear up the diagnosis.

In this connection it is important to mention, a point which is strongly emphasized elsewhere in this book (see p. 607), that symptoms of hyperthyroidism are very frequent in patients with incipient phthisis,

[1] Am. Rev. Tubercul., 1917, **1**, 289.
[2] Arch. d. Méd., 1919.
[3] Paris Méd., 1920, **10**, 80.

but that this indicates a good prognosis. In fact, it may be stated that hyperactivity of the thyroid is incompatible with progressive phthisis within certain limits. There is therefore no reason to make a hasty diagnosis in patients who cough, present some indefinite signs of an apical lesion, and incidentally show symptoms of hyperthyroidism. If the lesion is tuberculous, even when tubercle bacilli are found in the sputum, which is very rare, the outlook for recovery is excellent in the vast majority of cases, and the chances are that we are not likely to lose much by delaying treatment for tuberculosis.

There are good reasons for believing that the syndrome recently described as *neurocirculatory asthenia*, and which some believe to be one of the manifestations of hyperthyroidism, is also not likely to develop into phthisis.

CHAPTER XXIX.

COMPLICATIONS OF PHTHISIS.

MOST of the pathological processes described as complications of phthisis are part and parcel of the tuberculosis process, or symptoms of the disease which, at times, assume the ascendency. This is the case with hemoptysis, ulceration and amyloid degeneration of the intestines, tuberculosis of the larynx, kidneys, meninges, etc. Many of these conditions have been discussed while speaking of the symptomatology of phthisis. Pleural complications, such as pleurisy, pneumothorax, etc., are treated in special chapters. Here a few of the more important complicating processes, which may have an influence on the course or the prognosis of pulmonary tuberculosis, will be discussed.

Laryngeal Tuberculosis.—The frequency of this complication during the course of phthisis has been differently stated by various authors. The proportion varies from 5 to 50 per cent. Harold Barwell found at the Mount Vernon Sanatorium 11.69 per cent among 1541 tuberculous patients; Brandenburg, 9.16 per cent; John B. Hawes,[1] only 8 per cent among 1245 patients. Even sanatoriums, which do not admit patients with laryngeal complications, have many with this disorder. Thus at Otisville, N. Y., Julius Dworetzky[2] found that 25.6 per cent had laryngeal tuberculosis. Among 100 tuberculous children under fourteen years of age Dworetzky found no case of laryngeal tuberculosis. It seems that the proportion found depends on the zeal displayed by the laryngologists looking for it. Percy Kidd[3] found that 50 per cent of fatal cases of phthisis showed tuberculous laryngitis at the autopsy, and of these only 20 to 50 per cent were recognized during life. The estimate that one out of three patients with active and progressive phthisis has a laryngeal lesion appears to be correct.

Laryngeal tuberculosis spells phthisis; primary tuberculosis of this organ is so exceedingly rare as to constitute a medical curiosity. It is more frequent among males than among females, the proportion being, according to Morel Mackenzie, 2.7 of the former to 1 of the latter. The reason for this disparity is that men are altogether more liable to throat affections, probably because of the abuse of tobacco, alcohol, and exposure to irritation by dust at their occupations. It is also likely to be more severe in men than in women.

[1] Boston Med. and Surg. Jour., 1914, **171**, 19.
[2] Ann. Otol., Rhinol., and Laryngol., 1914, **23**, 835.
[3] Allbutt's System of Medicine, **5**, 210.

Symptoms.—These depend on the location of the lesion in the larynx. Those in whom the interior of the larynx is affected do not suffer as much as those whose trouble lies at the entrance of the larynx. The symptoms are few in number. Hoarseness is present in all in whom the interior of the larynx is affected, and it may be of various degrees, from mild tiring of the voice to complete aphonia. On the other hand, pain is more frequent when the entrance of the larynx, especially the epiglottis, is affected, while the voice may in these cases be retained quite well. The pain may be spontaneous, radiating to the ear, or there may be a sensation of tickling which provokes cough. In advanced cases, with perichondritis, deep ulceration of the epiglottis, and collateral inflammatory edema of the parts, the pain may be so severe as to interfere with swallowing food. Usually warm fluids and solids cannot be passed. The dysphagia may be so severe as to prevent

FIG. 106.—Tuberculosis of the larynx. (Ballenger.)

swallowing altogether. I have seen some cases in which swallowing of saliva was more painful than that of food. Local external tenderness is rare. Stridor, and obstruction of respiration, are comparatively rare, but they do occur now and then.

Julius Dworetzky, whose experience has been immense, classifies the clinical course of laryngeal tuberculosis into the acute, subacute, and the chronic types. The least frequent is *the acute type*, which is characterized by a soft edema of the larynx with a marked tendency to ulceration and no tendency to fibrosis. It is usually found in far advanced cases, but may, on rare occasions, be met with in incipients. Hoarseness, a sensation of fulness in the throat, dysphagia, etc., are very much accentuated. The outlook is grave; nearly all patients succumb within a few months. The *subacute type* shows a moderate tendency to fibrosis of the lesion. Papilliform infiltrates and soft

polypoid excrescences are usually found laryngoscopically. When the true vocal cords or the interarytenoid sulcus are involved, hoarseness is a clinical feature of these cases. The prognosis is favorable, especially if proper treatment is instituted. In *the chronic type* the tendency to

FIG. 107.—Incipient tuberculosis of the larynx. Infiltration of posterior commissure with slight thickening of arytenoids. (Dworetzky.)

FIG. 108.—Chronic tuberculosis of the larynx. Papillomatous infiltration of the posterior half of right cord with slight thickening at interarytenoid space. (Dworetzky.)

fibrosis and healing is strongly marked. The symptoms referable to the larynx are mild, or may be lacking altogether. The prognosis is excellent. It is this type of laryngeal tuberculosis which may exist for a long time without annoying the patient very much.

FIG. 109.—Chronic tuberculosis of the larynx. Left cord thickened owing to tuberculous infiltration; right cord slightly so. Slight interarytenoid thickening. (Dworetzky.)

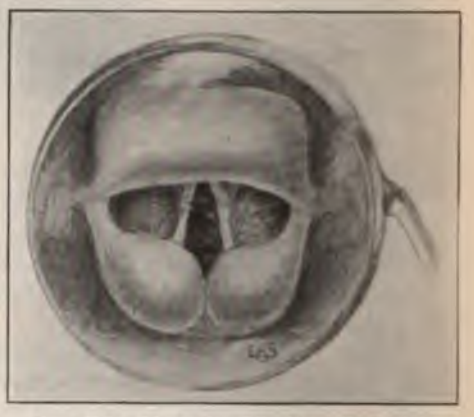


FIG. 110.—Marked infiltration of epiglottis; pear-shaped arytenoids. Infiltration and erosions of both false and true cords. (Dworetsky.)

Diagnosis.—Considering the immense prognostic significance of laryngeal tuberculosis, we must be guarded in making a diagnosis of this complication. *Hoarseness alone is insufficient for a diagnosis,*

because it may be absent when the larynx is implicated but the vocal cords remain in good shape; or it may be present in a patient suffering from phthisis, yet no tuberculous lesion is discoverable in the larynx. This is seen when the right recurrent laryngeal nerve is implicated in a thickened right apical pleural lesion, or when the two laryngeal nerves are pressed upon by enlarged tracheal glands. It must also be borne in mind that simple chronic laryngitis and pharyngitis are extremely common in phthisical subjects, as has been pointed out by Harold S. Barwell,[1] and they may cause hoarseness and throat discomfort. The constant coughing and the irritation of the sputum passing through the larynx may produce a simple laryngeal catarrh.

W. Freudenthal[2] urges that lasting hoarseness, apparently due to simple laryngitis, and seen in a patient who is not presenting symptoms of alcoholism or constitutional diseases, as gout or rheumatism, should excite suspicion of tuberculosis.

FIG. 111.—Far advanced tuberculosis of the larynx. Erosion of the entire right vocal cord; infiltration and erosion of right ventricular band. "Mouse-eaten" appearance of the left cord and hyperplasia of posterior commissure; infiltration of both arytenoids. (Dworetsky.)

FIG. 112.—Erosion of right half of epiglottis and right aryepiglottidean fold. Ulceration of right arytenoid. (Dworetsky.)

The diagnosis of tuberculous laryngitis is quite easy when there are ulcerations, but in the incipient stage it appears to be just as difficult as the diagnosis of incipient pulmonary tuberculosis. Laryngologists usually enumerate the laryngoscopic signs of advanced disease, evidently because they mostly see advanced cases.

Some authors have maintained that the tuberculous larynx is characterized by pallor of the mucous membrane. But it appears that pallor alone is insufficient for a diagnosis, because the larynx shares the pallor of the fauces which is seen in most tuberculous patients; it is also found in those who suffer from severe anemia of any kind. In fact, there are just as many red and congested larynges in phthisical subjects as pale ones.

[1] Lancet, 1909, **1**, 1249.

[2] Ztschr. f. Tuberkulose, 1910, **16**, 338.

Paresis of the vocal cord on the side of the lung lesion, associated with slight chronic laryngitis, is one of the signs of incipient tuberculosis of the larynx, according to many authors, notably F. Stern.[1] He calls this the "larynx sign" of early pulmonary tuberculosis and advises direct visual inspection to detect it when there is a sensation of vague oppression of the chest, a tendency to rheumatic pains, slightly irregular breathing, or gastric disturbances. The entrance to the throat is moderately red, and the paralyzed vocal cord is also red. There is always more mucus on the paretic cord than on the other, and its inner margin is usually irregular in outline. There is slight hoarseness, particularly at night, and the patient hawks often, but raises very little sputum, and tubercle bacilli may not be found at this early stage.

Thickening and even ulceration of the posterior wall of the larynx is another early sign. Uniform redness of both vocal cords is not pathognomonic of tuberculosis, but when one cord is red while the other remains normal or is pale, tuberculosis is probably present.

With the advance of the process the smooth and shiny appearance of the parts is changed owing to the ulceration. The infiltration often affects the epiglottis, producing that pale, rounded, sausage-like body which may attain such dimensions as to obstruct the view of the interior of the larynx. The arytenoid cartilages often change into pyriform bodies. When the infiltration begins to ulcerate, the characteristic worm-eaten appearance of the parts is seen, together with caries, perichondritis, necrosis, and exfoliation of parts of the cartilages.

In cases in which the infiltration begins in one or both vocal cords or the ventricular bands, or the interarytenoid region, the prognosis is more favorable. However, one or both cords may be destroyed by ulceration. In far-advanced cases all parts may be destroyed, including the epiglottis, of which only a short stump may be left.

Prognosis. — *The outlook in phthisis complicated by tuberculosis laryngitis is rather gloomy, though not invariably fatal, as was once thought.* Forty years ago Morell Mackenzie stated that "it is not certain that any cases ever recover." His statistics showed that it reduced the average expectation of life to twelve or eighteen months, very few patients living more than two and a half years. But since phthisis has decreased in malignancy during recent years, patients suffering from laryngeal tuberculosis have also benefited, and we now know that many recover. The lesion in the throat may heal, as has been found by careful studies of postmortem findings. St. Clair Thomson[2] recently reported a case treated twenty years ago, the patient keeping well ever since.

The laryngeal lesion *per se* only rarely kills the patient, and it has been stated that "consumptives never die from the larynx." This is wrong, of course, because we occasionally see a case of sudden death

[1] Berl. klin. Wchnschr., 1914, **51**, 1419. [2] Tubercle, 1919, **1**, 13.

from asphyxia or edema of the glottis. The bulk of the patients with laryngeal complication die as a result of the severity of the pulmonary lesion, or inanition, due to painful deglutition. In fact, when the larynx is extensively involved, producing dysphagia, dysphonia, etc., a fatal issue may be expected sooner or later. If the lesions in the lung and larynx are not sufficient to kill the patient he will die as a result of inanition.

The milder subacute and chronic forms of laryngeal tuberculosis have a better outlook. Many heal spontaneously without any local treatment. The general treatment instituted often hastens recovery from the laryngeal lesion. Very often the condition of the larynx goes hand-in-hand with the general condition of the patient, both improving, or aggravating, simultaneously. Very few are benefited by local treatment.

Gangrene of the Lungs.—This is an exceedingly rare complication of phthisis; it is more often found in cases of bronchiectasis, especially in old subjects. Considering that mixed infection is very frequent in phthisis, although the contaminating microörganisms are not responsible for most of the symptoms of the disease, it is surprising that putrefactive germs should but rarely take root in phthisical lungs. When occurring, it is soon recognized by the fetid breath and expectoration. But not all phthisical patients with fetid sputum have gangrene of the lung. Sputum retained in tuberculous cavities may become fetid. In such cases the malodorous expectoration lasts only for a few days or weeks, and sooner or later assumes the odor usually met in phthisis. Its odor also is different from that of gangrenous sputum —it is of a sweetish and nauseating character, while in gangrene it is pungent, and actually suffocating. The constitutional symptoms in gangrene are characteristic: The temperature rises high, the patient passes into a septic state with acute asthenia, and succumbs rapidly. In afebrile cases of phthisis a sudden rise in the temperature, accompanied by fetid sputum, is a sure indication of complicating gangrene of the lung.

In two cases of pulmonary gangrene complicating phthisis we have recently found various forms of spirochetæ and fusiform bacilli in the sputum; in one case these organisms were found in the gangrenous tissue after the lung was removed at the necropsy of the patient. As was the case in spirochetal gangrene of the lung without tuberculous lesions[1] these patients suffered from pyorrhea alveolaris. There thus seems to be justification for the assumption that the complicating gangrene is due to spirochetæ which are carried to the lungs by aspiration, or more likely by hematogenous metastasis of the infective material from the gums. On the other hand, we have recently also found spirochetæ in the sputum of tuberculous patients without gangrene of the lungs.

[1] Fishberg and Kline: Arch. Intern. Med., 1921, **27**, 61.

Tuberculous Ulceration of the Intestines.—The frequency of intestinal ulcerations found at autopsies on tuberculous subjects would indicate that they are more frequent than they are diagnosed *intra vitem*. Thus, Louis found ulcers in five-sixths of his cases; Bayle and Lebert, in two-thirds; Williams found at the Brompton Hospital postmortems in 81 per cent intestinal ulcerations of a tuberculous nature; and Percy Kidd found them in 71 per cent. While they are responsible for the diarrhea in advanced phthisis in most cases, in many the looseness of the bowels is due to the toxemia, the toxic substances in the blood being eliminated through the intestines, or swallowed sputum is the cause. Lardaceous disease of the intestines is very frequently responsible, while errors in diet, especially an excess of fat, or of milk, and particularly raw eggs, may induce diarrhea which is difficult to control.

There may be eight, ten, or even twenty, motions a day, expelling loose, dark, or chocolate-colored matter, exceedingly fetid, and it may contain small sloughs from the bowels. Quite often it is tinged with blood, but copious hemorrhages from the bowel are exceedingly rare. K. W. Lange,[1] looking for occult blood in the stools of tuberculous patients, found that tuberculous ulceration of the intestine may exist for a long time without giving rise to bleeding, and from his researches it appears that a negative result of a test for occult blood does not exclude ulceration of the intestine. John M. Cruice[2] says that when hemorrhage occurs it is of grave prognostic significance. The first case of this kind was reported by Tonnelle in 1829. In 1892 Guyenet could find only 15 cases in medical literature and Cruice found 10 additional cases in 1913. Although the prognosis is very grave in intestinal hemorrhage, L. S. Peters, Bullock, and Bonney report cases that recovered.

One characteristic of tuberculous diarrhea is its persistence. It may be checked by proper dietetic and medicinal treatment, but no sooner is this omitted than it reappears. With the diarrhea the emaciation proceeds at a rapid pace and they usually foreshadow quick relief from the suffering. I have seen patients who had been gaining, lose within one week all they gained in months, and within two to four weeks they were reduced to mere skeletons. But it must be mentioned that while diarrhea is a frequent symptom of tuberculous ulceration of the intestine, it is lacking in nearly 50 per cent of cases.

Diagnosis.—It is very difficult to say with certainty whether the diarrhea in a consumptive is due to toxemia or to intestinal ulceration. Tenderness is often found in the right iliac fossa, but it may be all over the abdomen, or any part of it. J. Walsh[3] made a thorough study of the symptomatology of intestinal ulceration, correlating it with autopsy findings in 100 cases at the Phipps Institute. The

[1] Ugeskrift for Laeger, 1917, **79**, 1371.
[2] Medical Record, 1913, **89**, 471.
[3] National Assn. Study and Prev. Tuberc., 1909, **5**, 217.

usual symptoms relied on—diarrhea and abdominal pains, tenderness and rigidity, especially in the region of the ileocecal valve—were carefully studied. He found that singly these symptoms add little or nothing to the diagnosis of intestinal tuberculosis, nor do any two, or all four when found in the same patient, because they may be encountered in cases in which the autopsy shows no ulcerations in the intestines, and the reverse. The presence of an ischiorectal abscess in an advanced case adds to the probability of intestinal ulcerations. Nor has he found any relation between the presence or absence of albumin or indican in the urine, or the results of the diazo-reaction, and intestinal ulceration. He concludes that the diagnosis of intestinal tuberculosis cannot be made with the slightest degree of certainty from our present known symptoms, and since the condition carries with it such an unfavorable prognosis, he advises that it is best that the diagnosis should not be made, so that the patient will have a better chance for hopeful treatment.

During recent years some authors have urged the *x*-rays in the diagnosis of intestinal tuberculosis. In 1912 Béclére and Mériel[1] showed that an ulcerated mucous membrane can be detected by a study of its motility; peristalsis was found abnormally strong, and the contents of the bowel were rapidly evacuated after reaching the colon. Similarly Stierlin[2] also found excessive motility of the contents of the colon in cases with tuberculous ulceration. Brown and Sampson,[3] repeating these investigations, arrived at the conclusion that when in a patient with pulmonary tuberculosis the roentgenologic picture shows hypermotility and spasm of the colon, or filling defects, a definite diagnosis of colonic tuberculosis may be made. A careful investigation of this problem in my wards at the Montefiore Hospital has not confirmed these findings. In cases with undoubted tuberculous ulceration of the intestines, verified by necropsy, no hypermotility of the colon or cecum was found, and filling defects were lacking, and the reverse.

While the outlook for healing of these ulcers is remote, yet it is possible. Amenomiya[4] shows that regeneration and healing are possible even without scar formation, but the muscular coat is never regenerated.

Peritonitis.—The pathogenesis of tuberculous peritonitis as a complication of phthisis is no more the disputed problem which it was formerly. Considering the frequency of bacilliemia in phthisis, it is clear that the blood may bring tubercle bacilli to the peritoneum just as readily as to other serous membranes. It is not so frequent a complication as is laryngeal or intestinal tuberculosis, but it apparently occurs more often than is suspected at the bedside, and we are at times surprised to find it at the autopsy when, *intra vitam*, even in carefully watched cases, it was not suspected.

[1] Congrès française de chirurgie, Paris, 1912, **25**, 103.
[2] München. med. Wchnschr., 1911, **58**, 1231.
[3] Jour. Am. Med. Assn., 1919, **73**, 77.
[4] Virchows Archives, 1910, **201**, 231.

Authors disagree as to its frequency in phthisis. Munstermann[1] found it in 5 per cent of cases; Borschke[2] in 16.17 per cent. In his autopsy material P. Horton-Smith Hartley found it in only 3.4 per cent of cases. Perforation of tuberculous ulcers of the bowels was observed in 3 cases out of 263 autopsies, or a percentage of 1.1, the perforation in each of the instances occurring in the ileum. It appears to be very frequent in acute miliary tuberculosis, but in chronic pulmonary tuberculosis it is less often encountered. While in many cases the infection of the peritoneum can only be explained by assuming that the bacilli were brought there by the blood, in a considerable number they may travel by way of the lymphatics from the pleura, the pericardium, from the mesenteric lymph glands and above all by contiguity from infiltrated Peyer's patches and ulcers of the intestines. They may also come by contiguity from tuberculous lesions of the urogenital system, especially from the adrenals, which are often the seat of tuberculous changes in phthisis.

Symptoms.—We meet mainly with two forms of this complication: dry, adhesive, and moist or exudative, both of which may be acute or chronic. During the course of phthisis the acute form, in the clinical sense, is usually due to perforation of an intestinal ulcer, or, more rarely, a pyothorax breaking into the peritoneal cavity, when it may produce suppurative peritonitis. In one case, in which during life the condition was not even suspected, I found at the autopsy a minute opening through the diaphragm permitting leakage of the pus from a pyopneumothorax. In such cases the symptoms are those of acute septic peritonitis. Fenwick[3] maintains that in some cases there may be premonitory symptoms, viz., pain for a few days before actual perforation takes place from a tuberculous intestinal ulcer; in others there may be bilious vomiting, the abdomen is distended, and hyperresonant on percussion. These premonitory symptoms are obviously due to local acute peritonitis. The actual perforation may occur during straining at stool, during an attack of vomiting or retching, or altogether while the patient is at rest. Some patients feel acute pain or a sensation as if something had given way in the abdomen. Collapse ensues and within a few hours or days the patient succumbs to cardiac failure. Some recuperate from the shock but they succumb within a few days to the symptoms of acute peritonitis, or more rarely to exhaustion.

The chronic form may be overlooked because it often runs its course symptomless. The patient may complain of abdominal pain, vomit, and have diarrhea, but these symptoms are very frequent during the course of phthisis without any peritoneal complication. On the other hand, there are cases with peritonitis in which these symptoms are lacking. The ascitic form is exceedingly rare in phthisis, though now

[1] Die Bauchfelltuberkulose, Munich, 1890.
[2] Virchows Archives, 1892, **127**, 121.
[3] Dyspepsia of Phthisis, London, 1894, p. 176.

and then we meet with a case in which the abdomen is filled with fluid. To be sure, there are many cases with exudates, but they usually escape detection until they assume large dimensions.

The fluid must be considerable to be discoverable by percussion. F. Mueller experimented on cadavers and found that in children under one year of age 200 cc of fluid in the peritoneum may be discovered in the peritoneum by percussion. In adults only two liters gave percussion signs; 1.5 liters gave some dulness in the dependent portions, while 1 liter could not be detected. In the living, Mueller, Sahli, and others state, conditions are more favorable, because of the elasticity of the abdominal wall and viscera. Small effusions may be detected in the knee-chest position. In many cases the patients note that while the chest and extremities become extremely emaciated, the abdomen grows in size.

The ascitic fluid may be serous, serofibrinous, hemorrhagic, and, rarely, purulent. In extremely rare instances it is found to be chylous. Tubercle bacilli are frequently found in it. Microscopically an excess of lymphocytes are found.

The adhesive form is characterized by the formation of adhesions and cicatricial contractions of the mesentery and gluing together of loops of the gut are very frequent. Especially frequent are adhesions of the peritoneum to the liver and spleen. The adhesions and cicatricial contractions, at times, produce incomplete stenosis of the intestine with resultant persistent constipation and uncontrollable vomiting. Colicky pains, increased by pressure and on movement, may be observed. In these cases the emaciation may be extreme, despite the fact that the local lesion in the lungs is not extensive nor very active. When the inflammation in the peritoneum is limited and circumscribed, which is not infrequent, the pain may be localized at one point. It is noteworthy that fever may be absent, but in most cases of active phthisis, pyrexia due to the lung lesion is so frequent that it cannot be utilized for diagnostic purposes as to the presence or absence of a peritoneal complication. On the other hand, when the lesion in the lung is quiescent or latent, the complicating peritonitis may pass an apyretic course. In many cases there is diarrhea due to intestinal catarrh or, more frequently, to ulcerations of the intestine.

As was already stated, many cases run their course painlessly. When copious, the exudate is easily detected by the usual physical signs bearing in mind that the fluid is shifting with the position of the body. In others it is encysted because of plastic fibrinous formation. Thormayer[1] described physical signs which he considers characteristic of tuberculous and carcinomatous peritonitis. He found that tympany is very frequently elicited on the right side of the abdomen, while on the left side a dull note is elicited by percussion. He explains this phenomenon on anatomical grounds: The mesentery in the right

[1] Ztschr. f. klin. Med., 1884, **7**, 378.

side usually contracts more than in the left, and thus intestinal coils are apt to be drawn to the right by the shrinking mesentery; tympany is then elicited over these distended intestinal coils. It is, however, an inconstant symptom and if it occurs at all, it is discerned late, after the organization of the exudate.

At times we may, on palpating the abdomen, feel some crepitation, and in some cases I have even heard friction sounds while auscultating with the stethoscope. In rare instances, tumor-like masses are palpable in the abdomen. When localized in the right side they may simulate appendicitis. In one case under my care repeated attacks of pain in the right lower part of the abdomen, constipation, and even rigidity of the rectus muscle exquisitely simulated appendictis. But later when a tumor was palpable the condition was cleared up. In another case under my care symptoms not unlike those of intestinal obstruction were present in a woman with tuberculous pleurisy, and the advisability of operative interference was seriously considered, but the patient recovered. It appears that tuberculous cicatrices causing narrowing ot the gut may stretch, and thus relief ensues. This is also true of cicatrices of the intestinal wall caused by healing tuberculous ulcers.

Cardiac Complications.—We have shown that phthisis only exceptionally develops in persons suffering from chronic endocarditis, excepting in those with congenital heart disease (p. 127). But endocarditis may develop during the course of phthisis, either due to complicating rheumatic disease, or any other accidental septic process, as tonsillitis, etc. The verrucose excrescences on the cardiac valves often found at autopsies on phthisical subjects are usually caused by other microörganisms, though Heller, Leyden, Benda, Tripier, and others maintain that tubercle bacilli may be responsible in some cases.

Myocarditis.—In most cases heart failure in advanced phthisis is due to myocarditis, with dilatation of the right heart; to tuberculous pericarditis with adhesions to the pleura, and also to dilatation with cardiac displacement. Like in other chronic, cachectic, and exhausting diseases, the myocardium partakes in the atrophy of the muscular system and gives way from sheer exhaustion. In fibroid phthisis, and the pleural forms of chronic phthisis, the induration in the lungs interferes with the circulation, and heart failure of variable degree is the result. Before the onset of decompensation, hypertrophy of the right ventricle is quite common, especially in fibroid phthisis.

Pulsations in the epigastrium, and accentuation of the second pulmonic sound reveal this condition. However, accentuation of the second pulmonic sound may be present without hypertrophy when the left lung is retracted through infiltration or shrinkage and reveals the left heart. The constitutional symptoms of heart failure—dyspnea, edema, etc.—may be quite marked.

Pericarditis.—Pericarditis may occur during the course of chronic phthisis. Several cases of primary tuberculous pericarditis have been

reported. In chronic phthisis the pericardial sac may be implicated by tuberculous processes of the pleura or mediastinal glands. Adhesions between the pleura and pericardium are often found and with the shrinkage of the affected lung the heart is pulled out of its normal position, as has already been described.

Very often we meet with acute pericarditis in phthisis and pleuropericardial friction sounds may be audible. The symptoms and signs characteristic of adhesive pericarditis are not rare in chronic phthisis—systolic retraction of the chest wall at the apex, engorgement of the veins in the neck, disappearance or weakening of the pulse during inspiration, etc.

In very rare instances we meet with acute pericarditis coming on suddenly with pain in the cardiac region, dyspnea, cyanosis, cardiac irregularity, etc. In one case under my care the symptoms simulated pneumothorax. Careful examination of the heart, however, clears up the case. The cardiac dulness is increased, friction sounds are audible, the apex beat disappears with the effusion. The pericardium may also be implicated in cases of pneumothorax, producing pneumopericardium, as has already been mentioned.

Phlebitis and Thrombosis.—Although occurring quite frequently during the course of phthisis, phlebitis and thrombophlebitis are only rarely mentioned as complications of this disease. Older clinicians, as Hoffmann in 1740, and after him Hunter, Louis, Trousseau, and others have mentioned it, and Cursham wrote in 1860 on "Causes of Obstruction of the Veins of the Lower Extremities Causing Edema of the Corresponding Limb and Occurring in Phthisical Patients." Most writers are inclined to attribute them to the tuberculous toxemia, while others have found in them an instance of marantic thrombosis. But Gustav Liebermeister,[1] in a thorough clinical and pathological study of the subject, attributes them to the direct action of the bacilli on the bloodvessels, finding as he does that nearly all tuberculous patients have a bacteremia. Haushalter and Etiénne, Vaquez, Sabrazés and Mongour, Chantemesse and Widal, Lesné and Revaut, Liebermeister, and others have found virulent tubercle bacilli in such thrombi. In cases under my care no tubercle bacilli could be found in the thrombi microscopically or by inoculation of animals.

Phlebitis and thrombosis in phthisis usually occur in the femoral vein, though at times we meet with cases in which the vena cava, the innominate, jugular, subclavian, or renal veins or even the cerebral sinuses are affected. The frequency of this complication is given by P. R. Dowdell[2] as 30 among 1300 consumptives, or 1.5 per cent. H. Ruge and Hierokles[3] found it 19 times among 1778 cases of pulmonary tuberculosis, or 1 per cent. In my experience it appears to be even more frequent in advanced and active cases of phthisis.

[1] Virchows Archiv, 1909, **198**, 332.
[2] Am. Jour. Med. Sci., 1893, **105**, 641.
[3] Berl. klin. Wchnschr., 1899, **36**, 73.

P. Horton-Smith Hartley found thrombosis of veins in 2.6 per cent of 263 cases which came to autopsy. In males the percentage was but 1, while in females it was 6.6. Ethan A. Gray observed thrombophlebitis as a complication of phthisis 7 times in 1400 cases at the Chicago Fresh Air Hospital: 3 in men and 4 in women.

Phlebitis is at times found in the veins of the upper or lower extremities, especially in very active cases running high fever. Mostly the medium-sized or small veins are affected. Clinically, the thickening of the veins of the upper extremities are more easily recognized by palpation because of the lesser thickness of the muscles and adipose tissue. The affected veins are tender to the touch and also painful on motion of the limb. Edema of the extremities is exceptional in simple phlebitis, though in some cases it may occur. The phlebitis may disappear, to reappear again, and in most cases it is persistent till thrombosis also occurs, or till the fatal issue of the case. In fact, *phlebitis is an ominous complication.* A thrombus may develop and it may soften and be carried by the circulating blood to distant organs, producing pulmonary embolism or infarction. It may organize and remain as a firm, thick cord. Hirtz[1] described cases of phlebitis and thrombosis occurring during the incipient stage of phthisis, or even preceding the actual onset of the disease, especially in chlorotic girls.

Thrombosis of the Femoral Veins.—Thrombosis occurs most frequently in the femoral vein but, as was pointed out by Dowdell, usually the popliteal vein is found to contain a clot of older date, while in some the saphenous vein is plugged and rarely the superficial veins of the leg and thigh, as well as the main trunk from the tibial vein upward, are thrombosed. Dowdell, Ruge and Hierokles, Liebermeister, and others have also found thrombosis of the uterine and brachial veins, the prostatic plexus, and embolism of distant arteries is said to be not uncommon. As is the case with phlebitis, thrombosis is found mostly in far-advanced but acutely running cases and is usually the precursor of a fatal issue.

The most important symptom is edema of the affected limb. The onset is usually slow and insidious, the swelling coming on gradually. Pain is often felt for a few days after the onset of edema, but in many cases this is lacking. When present it is mainly felt in the popliteal space where tenderness may be elicited. Inasmuch as practically all these patients have symptoms of active phthisis, the temperature is not an aid in the diagnosis—it is continuous or hectic, as the case may be; the onset of the thrombosis, edema, etc., has hardly any influence on the pyrexia. In some cases under my care there were disturbances in sensation of the affected limb, which was cold, numb, or tender. In one case the pain was excruciating and morphine alone was effective in relieving it in part. When the deeper veins of the muscles are plugged, which is not rare, there may be severe pain and hyperesthesia of the calf of the affected leg. Diagnosis may be difficult

[1] Semaine Médicale, 1894, **14**, 274.

at first, but as soon as the edema appears, the cause is clear. In some cases the thrombus in the affected vein is so thick as to be palpable. I have many times been able to palpate the femoral and popliteal veins as thick, firm cords tender to the touch.

Diagnosis.—In most cases the diagnosis of thrombosis and phlebitis is rather easy. It is to be differentiated from edema of the extremities common in phthisis and due to cardiac and renal insufficiency, and from cachectic edema which is frequently seen in the terminal stages of this disease. Thrombosis always begins in one extremity and is confined to it, or marked on one side when fully developed. It is tender to the touch along the course of the veins and not necessarily over the edematous skin. The dilated superficial veins may at times contain clots. On the other hand, edema due to cardiac or renal disease is accompanied by signs and symptoms of these conditions, both lower extremities are affected by the swelling, and the tenderness along the course of the veins is lacking. Cachectic edema occurs on both sides, is painless and subsides when the patient is kept in the recumbent position for some time. At times intra-abdominal pressure by enlarged mesenteric glands, or bands in adhesive peritonitis, on the common or external iliac vein, or on the femoral may produce edema of one extremity not unlike that of thrombosis. The same condition may occur, though very rarely, in the upper extremity when intrathoracic pressure is exerted by enlarged glands in the thorax on the main trunks of the veins. But careful examination will usually reveal the tumor or the glands which are responsible.

Thrombosis of the Jugular Vein.—Thrombosis causing edema of the upper extremity is very rare, but it does occur. Four cases have come under my observation. Humphrey[1] reported such a case in 1859; Lésague[2] observed in 1870 a case of phthisis complicated by the formation of a thrombus in the external jugular, subclavian, and humeral veins. Ten days after the appearance of the thrombus it was completely softened and all symptoms of phlebitis disappeared. But in all other cases reported, death supervened within a couple of weeks after the establishment of thrombosis. The symptoms are edema, pain, etc., of the upper extremity. In 1904 Charles J. Aldrich[3] collected from the literature 9 cases of this complication of phthisis and reported 1 of thrombosis of the left internal jugular with extension through the subclavian down the axillary into the basilic veins. Two weeks later a like thrombus appeared in the right side and extended to the veins of the arm. Death was due to cerebral sinus thrombosis from extension of the thrombus in the right internal jugular vein. In one of my cases thrombosis of the right internal jugular vein occurred in a patient with pneumothorax.

Prognosis of Thrombosis.—The prognosis is fatal in the vast majority of cases because of the severity of the tuberculous process, occurring as

[1] British Med. Jour., 1859, 582, 601, 619, 650.
[2] Gaz. Méd. de Paris, 1879, **1**, 649.
[3] New York Med. Jour., 1904, **79**, 442.

it does mainly in rapidly advancing cases of phthisis. Death may be due to secondary emboli which is then sudden. But I have recently seen several cases in which the symptoms of phlebitis and even thrombosis have cleared up and the phthisical disease went on its course. Lésague and Ethan A. Gray have also reported cases of this character.

Urogenital Tract.—Of other complications occurring more or less often during the course of phthisis may be mentioned tuberculosis of the urogenital tract. We have already mentioned that albuminuria is not uncommon in phthisis. In far-advanced cases, nephritis is quite frequent and we may have most of the symptoms of this disease, especially edema, etc., and even uremia, which is at times difficult to differentiate from tuberculous meningitis. In many of the advanced cases we may also note symptoms due to amyloid disease of the kidneys: Abundance of secretion of urine of low specific gravity containing hyaline and especially waxy casts and albumin in large quantities. But in this form of nephritis dropsy is infrequent. I have been struck with the fact that in most cases in which there is considerable albumin in the urine and dropsy, the temperature drops down to near normal and very often the activity of the process in the lung diminishes. The prognosis is, however, not improved. On the other hand, chronic nephritis, when complicated by pulmonary tuberculosis has a rather favorable influence on the lung condition (see p. 604).

In some cases *tuberculosis of the kidneys* supervenes and also of the bladder, seminal vesicles, vas deferens, and epididymis. Tuberculosis of the kidneys is very difficult of diagnosis in its early stages. Finding acid-fast bacilli in the sediment of the urine is not sufficient to base a diagnosis in my experience, excepting when the specimen has been obtained by catheterization of the ureter. Even so there have been reported cases in which tubercle bacilli were found microscopically and by inoculation into animals, yet the autopsy, or the kidney removed by operation, showed no tuberculous lesion. This is a fact which should never be lost sight of in doubtful cases. I have seen cases in which tubercle bacilli were thus found yet the patient improved without operation. Patients with *tuberculous pyelitis* suffer usually from lumbar pain of a dull character, have pus, albumin and blood, epithelium, and even caseous débris in the urine. At times there are seen cases in which the pain occurs in paroxysms and it is difficult of differentiation from that of renal colic due to stone. The reciprocal relations between renal and pulmonary tuberculosis are discussed elsewhere (see p. 587).

Terminal Edema.—In a large proportion of tuberculous patients edema, general or local, appears a few days, or weeks, before the fatal termination of the case. The edematous swelling is mainly seen around the joints of the lower extremities; but at times it involves the whole body. The origin of this edema is not known definitely. Some are inclined to attribute it to nephritis, but it is met with in cases in which the autopsy shows that the kidneys remained in good condition.

Others state that it is due to myocardial degeneration, especially to dilatation of the right ventricle. Charles W. Mills[1] and John T. Henderson found a characteristic picture by Mosenthal's test, with a marked decrease in water and sodium chloride elimination.

Tuberculous Ulcerations of Mucous Membranes.—We have already pointed out that despite the fact that so much of tuberculous sputum passes through the mouths and lips of phthisical subjects, ulcerations of these parts are extremely rare. But it appears that tuberculous ulceration of the tongue is more frequent than is generally appreciated. Jame R. Scott has recently drawn attention to this fact. At the Montefiore Hospital I see about eight or ten cases a year. These ulcers may appear fissured, granulomatous, or papillomatous; in many cases they are located on the dorsum of the tongue but very frequently also on the tip, the sides and, rarely, on the frenum. I have seen some with ulcers of the soft palate, and very rarely on the posterior wall of the pharynx. In a case under my care there were three ulcers on the tongue, one on the tip and two on each side.

In most cases the diagnosis is clear, occurring as they do in patients with pronounced tuberculous lesions in the lungs and perhaps the larynx. But, at times, they may be found in a patient without very active symptoms of phthisis, and must then be differentiated from local manifestations of syphilis, carcinoma, and epithelioma. A careful examination of the chest will clear up the diagnosis, because these ulcerations are, almost without exception, secondary to tuberculosis in the lung. A specimen removed and examined microscopically may show the characteristic tuberculous changes or tubercle bacilli.

Purpura.—I have seen several cases of purpura hemorrhagica complicating advanced phthisis. Petechia are very frequent in many cases, but true purpura hemorrhagica with extensive ecchymoses scattered over the limbs may occur, and there may be simultaneously hemorrhages from some of the mucous membranes—true purpura hemorrhagica. In 3 out of the 4 cases seen by me recently there were also albuminuria and hematuria, and the patients succumbed shortly after the appearance of the purpura, and I am inclined to agree with John M. Cruice[2] to the effect that the occurrence of purpura especially the hemorrhagic form, in the course of tuberculosis is always a grave symptom.

Its etiological relation to tuberculosis is doubtful. Some authors are inclined to see in the tubercle bacillus a cause of the purpura, but the fact that it is so extremely rare in phthisical subjects shows that when the two diseases occur in the same subject, it is in all probability a coincidence. I believe that Cruice's observation that after an attack of purpura physical examination will reveal a more advanced condition of the lesion does not at all prove that the hemorrhages into the skin were directly of a tuberculous character; it by no means excludes the chances of their being a coincidence.

[1] Am. Rev. Tuberc., 1917, **1**, 573.
[2] Am. Jour. Med. Sci., 1912, **144**, 875.

Superficial Cold Abscesses in the Chest Wall.—Though these abscesses are not very uncommon in tuberculous subjects, they are only rarely mentioned in monographs on the subject of tuberculosis. Their relation to phthisis was first pointed out by Leplat in 1876. Other French authors, notably Goujot, Duplay, Verneuil, Charvot, and others then described them in detail. Three varieties have been mentioned, one arising from the cellular tissues, one from the periosteum of the ribs, and a third of deep origin from the bone. Goujot described these abscesses as in front of the ribs, behind the ribs, and of the shirt-stud variety, in which a superficial and deep abscess communicates through an intercostal space.

S. Souligoux, Peron, Villar, Paget, and more recently Samuel Robinson,[1] show that these abscesses are of pleuropulmonary origin. Robinson, with considerable *x*-ray experience, shows that "the time-worn custom of regarding such lesions as due solely to a necrotic rib is unquestionably a fallacy." Some authors state that erosion and even necrosis, particularly of the posterior surface of the rib, are not uncommon, but this is purely incidental. Iselin,[2] who studied many cases is of the opinion that the ribs are involved only secondarily. In 98 operative cases, sequestra, or a probably primary bone lesion, were found only in 6 cases, and the ribs were involved only in 3 of them, and in 1 of these the patient had had syphilis. It usually follows old tuberculous pleurisy, but may be found in pulmonary cases. The tubercle bacilli apparently invade the chest wall through the lymphatics which may be found in old adhesions of the pleura. These abscesses have been found in rare instances, to drain by breaking through a tuberculous lung. They are analogous to the abscesses found often in the vicinity of the incision for empyema of tuberculous origin.

On the chest wall, along the line of insertion of the diaphragm, particularly anteriorly, or in the lower axillary region, there is noted a circumscribed swelling, the size of a pigeon's or a hen's egg, painless and fluctuating. I have recently seen cases in which the diameter of the abscess was five to six inches. There is usually no surrounding inflammatory induration, and only later the infected area becomes red and somewhat tender. When incised a moderate amount of liquid, curdy, pus is eliminated, but healing is slow: In most cases a fistula is left which persists for months; or an ulcer remains which keeps on discharging pus for a similar period. Very often the fistula or ulcer is located over a rib, the periosteum of which is implicated. In many cases healing finally takes place leaving an ugly red scar.

The diagnosis is at times difficult—there is a question whether it is not an empyema pointing on the chest wall, particularly when there are physical signs of a lung lesion or thick pleura elicited in the same area. A careful consideration of the history and course of the trouble, however, clears up the diagnosis.

[1] Tr. Nat. Assn., Study and Preven. of Tuberc., 1917, **13**, 170.
[2] Corr.-Bl. f. schweiz. Aerzte, 1919, **49**, 97.

CHAPTER XXX.

RECIPROCAL RELATIONS BETWEEN PULMONARY TUBERCULOSIS AND CERTAIN PHYSIOLOGICAL AND PATHOLOGICAL PROCESSES.

PULMONARY tuberculosis is a disease occurring at all ages and, in its most common form, the chronic, lasts for many years during which the patient is subject to the diverse physiological and pathological processes peculiar to mankind. Certain physiological processes, as was already shown elsewhere, have a great influence on the pathogenesis, course, and outcome of tuberculous infection and disease; others are in themselves greatly affected by the tuberculous toxemia. In the long months, or years, during which the patient suffers from the symptoms of phthisis, he is liable to contract other diseases which may have an influence on the symptomatology and course of the lung disease; or conversely, the intercurrent disease may be altered in its clinical manifestations because of the underlying tuberculous process. These reciprocal relations between pulmonary tuberculosis and physiological and various pathological processes are of immense importance to those who have tuberculous patients under their care. They have great bearings on the origin, diagnosis, prognosis, and treatment of the tuberculous patient. A knowledge of these interrelations is especially needed by those whose practice is limited to institutions devoted exclusively to the treatment of tuberculosis, where physicians are liable to become "tuberculosis specialists," oblivious of the fact that their patients are humans—liable to suffer from diseases other than that caused by the tubercle bacillus, and that an intercurrent disease may have an influence, at times a very favorable one, more frequently the reverse, on the tuberculous process.

Growth.—Tuberculosis attacks human beings at all periods of life. When the disease is active during the formative age, it usually retards physical development, stunts growth, leaves its victim with small, feeble, and flabby muscles, rather slim bones, and a notoriously deformed chest. As was already stated, the phthisical, or paralytic, chest is almost invariably the result of tuberculosis of the intrathoracic glands during infancy and childhood (see p. 303). It is a well-known fact that tuberculous children are puny, shorter than others, and weakly; even when they are fat, "pasty," which is not uncommon, their atrophied musculature betrays trouble. It is noteworthy that many of these undersized children begin to grow in height after twelve or fourteen years of age, and may become taller than the average for their

race stock. It seems that many of these tall youths present signs of dyspituitarism, such as prognatism, long extremeties, enlarged wrists and fingers, etc. Whether these changes are due to tuberculous changes in the pituitary, or to toxic effects on this gland, has not been established. It is, however, a fact commonly observed, that while these youths grow in height rapidly, at that age, certain secondary sexual characters are apt to be rather slow in their appearance; The axillary and pubic hair may be scanty or absent, the mammary glands in girls rather small, or rudimentary, menstruation delayed, etc. Stigmata of infantilism are common in young tuberculous subjects. At this age a smouldering tuberculous process in the intrathoracic glands is likely to flare up and invade the lungs. When this occurs, the prognosis is, as a rule, serious in the writer's experience. (See p. 610).

On the whole, it may be said that tuberculosis retards growth in height when attacking children under twelve years of age; it enhances it, when attacking adolescents between fourteen and twenty, and it has no effect on stature of adults in whom growth of the skeleton is an accomplished fact, unless it is accompanied by kyphosis or scoliosis, when it is reduced. Deformities of the chest occur in children as a result of tuberculosis; in adults they are seen to result from pleural adhesions and destruction of lung tissue, and manifest themselves in the form of flattening and localized retractions. This has been discussed in detail in Chapter XIV.

When judging the weight of children suspected of, or actually suffering from, tuberculosis the facts about growth are to be borne in mind. A youth under eighteen years of age does not hold his own if he does not gain in weight; the gain in height should be registered on the scale, otherwise there is an actual loss in weight. Reports of institutions caring for tuberculous children at times forget this factor of growth, and when they indicate that their little patients had gained in weight, they must show that this gain has been more than could be accounted for by the gain in height which the children have attained during the given period.

Puberty.—The interrelation between the respiratory and the generative organs in women is more intimate that is generally appreciated. During menstruation and pregnancy the mucous membranes of the nose and larynx are very often congested, and subject to catarrhal changes with or without visible causes. Changes in the voice are often noted in women during menstruation, pregnancy, the menopause, etc. For centuries clinicians have noted certain influences exerted by tuberculous disease on ovarian function.

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The tuberculous woman with amenorrhea may be recognized at first sight in most cases. The facies of tuberculosis (see p. 299) is rather sharply accentuated, and pallor of the face and extremities, which is not very common in the average case of tuberculosis, is more or less severe. We have shown that the blood picture in tuberculosis is not characteristic (see p. 281), but in women with scanty or absent menstruation, a blood count of secondary anemia is the rule.

Vicarious menstruation has been spoken of by many writers of former generations; hemoptysis of various degrees supplanting the periodical uterine flow. Careful clinical observation has, however, shown that in these cases we deal with ordinary tuberculous hemoptysis in phthisical women in whom amenorrhea is one of the cardinal symptoms. In some cases there is a tuberculous lesion which has not been diagnosed. I have never been satisfied that any of the cases that came under my observation could be considered genuine vicarious menstruation in the sense given it by some authors.

Menstrual hemoptysis, which has already been discussed (see p. 247), is observed at times. The patients spit blood during the menstrual period, or a few days preceding it, irrespective of the effect exercised by the tuberculous process on the ovarian function. This goes hand in hand with the liability of women to suffer from acute exacerbations of the tuberculous lung lesion at that period, manifesting itself in elevation of the temperature, increase in the severity of the cough, and expectoration, etc. The liability to congestions of the mucous membranes of the upper respiratory tract during pregnancy may be considered an analogous phenomenon. Similar effects of the menstrual function on a pathological process have been observed during the recent pandemics of influenza.

When occurring in the very young, tuberculosis often hinders the development of the primary and secondary sexual characters. The infantile type of uterus is common in young tuberculous girls; hypoplasia of the testicles in boys. The secondary sexual characters, the breasts, the pubic hair (also that in the axillæ) are often rudimentary, and the onset of menstruation may be delayed, as has already been noted.

Primary tuberculosis of the generative organs is very rare, though in fatal pulmonary phthisis specific tuberculous changes are frequently found in the female pelvic organs; some authors, like Schlimpert and [illegible]mig, have found this to be true in over 80 per cent of fatal cases of [illegible]ary tuberculosis. This is not in agreement with the results of made of my own material.

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race stock. It seems that many of these tall youths present signs of dyspituitarism, such as prognatism, long extremeties, enlarged wrists and fingers, etc. Whether these changes are due to tuberculous changes in the pituitary, or to toxic effects on this gland, has not been established. It is, however, a fact commonly observed, that while these youths grow in height rapidly, at that age, certain secondary sexual characters are apt to be rather slow in their appearance; The axillary and pubic hair may be scanty or absent, the mammary glands in girls rather small, or rudimentary, menstruation delayed, etc. Stigmata of infantilism are common in young tuberculous subjects. At this age a smouldering tuberculous process in the intrathoracic glands is likely to flare up and invade the lungs. When this occurs, the prognosis is, as a rule, serious in the writer's experience. (See p. 610).

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A large proportion of tuberculous women, especially adolescents, menstruate scantily; in many complete amenorrhea occurs for months. Instead of the sanguineous flow, there may be merely an increase in the amount of mucous discharge lasting for a day or two. In fact, amenorrhea without any assignable cause has been considered of tuberculous origin by physicians and the laity; while others have

considered it a cause of consumption. In a small proportion of cases menorrhagia or metrorrhagia is observed; in others, dysmenorrhea; several authors have claimed that this sort of painful menstruation may be relieved by proper antituberculosis treatment, especially tuberculin.

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excepting in fatal cases of phthisis, the lungs do not show much evidence of active disease in patients in whom the process in the generative organs is so pronounced and disabling as to require surgical intervention. Of the many cases of surgical tuberculosis of the female generative organs seen by the writer, patients in whom destructive tuberculous lesions have been demonstrated in the organs removed surgically, only few showed symptoms and signs of active tuberculous lesions in the lungs. This is also to be observed in many cases of tuberculosis of the testicles. It appears to be in agreement with our other observations that extrathoracic tuberculous lesions shield the lungs against active disease (see p. 584). Several cases have been observed in which dormant lung lesions were reactivated soon after surgical operations on the generative organs for tuberculous disease.

Sexual Function.—The popular views entertained by the laity and the profession to the effect that consumptives have excessive sexual potency and demands are apparently well founded. During the incipient stage of the disease there is often noted an increased sexual irritability, and this is apparently the reason why some believe that phthisis is at times due to excessive venery. Létulle asserts that sexual excesses are common at the commencement of the disease, and are checked only when the limit of exhaustion is reached. W. H. Peters[1] observed a tendency to abnormal sexual excitement so frequent among consumptives as to require careful attention of the physician. He says that "every physician has been impressed by the almost disgusting, and sometimes revolting persistence of the sexual instinct in consumptives, even late in the disease."

It is remarkable that in the advanced stages of the disease, when the body is extremely emaciated, the muscles atrophied and the vital forces apparently at their lowest, sexual potency may be retained. Even shortly before death a consumptive may impregnate his wife, and a woman who has lost half her normal weight, and is subject to frequent hemorrhages, runs a febrile temperature, sweats, and coughs distressingly, is, at times, seen in a pregnant state. Peters quotes Barnes, Superintendent of the Rhode Island State Sanatorium, about a patient who died from hemorrhage coming on during the sexual act which took place while on a visit from the sanatorium to his wife. I have met similar cases. In hospitals for advanced consumptives the patients must be watched in this regard, especially when the male division is not completely separated from the female division. Sexual excesses, according to Gimbert,[2] often hastens the fatal outcome of the disease.

Weygandt[3] made a collective investigation of this problem among physicians in German Santoriums in which incipient cases are admitted. Many of the answers were to the effect that they had not observed any

[1] Jour. Am. Med. Assn., 1908, **1**, 938.
[2] Rev. de la Tuberculose, 1907, **11**, 1.
[3] Med. Klinik, 1912, **8**, 91 and 137.

special increase in the sexual desires of their patients; three directors of sanatoriums, Kohler, Krause, and Marquard, sent the interesting information that the patients had accused the doctors of secretly putting aphrodisiac or anaphrodisiac drugs into the milk, or other food. It appears that in many German sanatoriums such superstitions prevail, thus indicating that the patients themselves are aware of the increased sexual irritability.

These sexual excesses have been attributed to the tuberculous toxemia. It has also been said that the lazy, indolent life, the lack of muscular exercise, and the excessive consumption of nitrogenous food during the treatment, are more responsible for the sexual proclivities than the tuberculous toxemia. Some consider the association of the sexes in sanatoriums favors tendencies in this direction. In many cases the despondency engendered by the knowledge of suffering from an incurable disease urges the patients to take in as much of life and its pleasures as possible before it is too late.

There are other chronic diseases in which the patients are idle, eat well and may become despondent, yet they do not indulge in sexual excess to the same extent as the tuberculous, which would be in line with the suggestion that the tuberculous toxemia is effective in the direction of causing sexual irritability. Turban found that in artificial tuberculin poisoning, *i. e.*, when tuberculin is administered for therapeutic purposes, sexual irritability is increased, and in some cases he had to discard specific treatment for this reason. "Every physician with extensive experience with tuberculous patients," says Muralt, "knows of cases in which recovery from the disease brought about normal functions in this regard."

Fecundation.—It is noteworthy that tuberculosis appears to have no influence on the function of fecundation, so long as the process in the lungs is not so far advanced as to render the patient completely bedridden. Most married tuberculous women become pregnant, unless measures are taken to prevent it. In fact, physicians with extensive experience among these patients have seen emaciated consumptive women, having fever, severe cough and profuse expectoration, hardly able to move about because of atrophy of the subcutaneous fat and muscles, become pregnant and, what is astonishing at times, they go on to term, and are delivered of full sized infants. Properly collated statistics on this point are not available, as far as the writer knows, because it is difficult to take into consideration the factor of prevention of conception, separation of the mates, etc. (See p. 298).

Pregnancy.--Considering their vast prophylactic bearings, the problems of the reciprocal relations between pregnancy and tuberculous disease have been widely discussed, though it cannot be said that they have been satisfactorily studied. For centuries many physicians stated that they had observed that pregnancy often ameliorates the condition of female tuberculous patients, and some, like Cullen (1712–1790), recommended marriage for tuberculous girls for this reason.

Dr. E. Warren,[1] in a prize essay published in 1857 said: "Pregnancy, coition, etc., are particularly desired by women affected with phthisis, which constitutes a *pointing of Nature* toward a *remedy* for the evils by which the system has been invaded." He quotes the opinion of authorities, like Hippocrates, Sydenham, Montgomery, Parr, Rokitansky, Clark, and many others, who held similar views on the effects of marriage and pregnancy on tuberculosis. There are writers who, at present, speak in the same vein: Thus, Charles W. Townsend,[2] speaking of cases observed in the Boston Lying-in Hospital, says that "during pregnancy the patient often seems better, and the disease appears in abeyance," and that "Nature seems to put forth a supreme effort to suppress the disease during pregnancy and make labor easy and short, but after the child is born the disease advances at a rapid rate. A French author, Sabourin,[3] is even more explicit in his recommendations of marriage for tuberculous girls because it seems to endow them with a certain degree of immunity and deprives them of the "catamenial intoxication, the greatest enemy of tuberculous women." A Spanish author, Lasbennes,[4] brings statistics showing that women of the child-bearing age have a lower mortality from tuberculosis than men, and concludes that matrimony is advisable for certain "pre-tuberculous" cases.

The vast majority of modern writers are, however, opposed to these views, and maintain that pregnancy, labor, and lactation are liable to reactivate latent tuberculosis, and aggravate active lesions, often leading to a fatal termination of otherwise favorable cases. Statistics in support of these contentions are not wanting. Some authors, like von Ysendick, Amstel, Rosthorn, and others state that this is the invariable outcome when a tuberculous woman becomes pregnant, that 100 per cent are either aggravated, or succumb; Pankow and Küpferle, have found this to be the case in 95 per cent of cases; Neltner, Kaminer, Deibel, Felner, Schauta, Eich, Pradella, and Reiche, between 60 and 90 per cent; Freund, in 38 per cent, while others calculated smaller percentages of aggravation and deaths, some even as low as 16 per cent (Norris). Likewise, the number of deaths because of aggravation of the disease soon after the puerperium oscillates within wide limits according to published statistics. Amstel reports 100 per cent; Lebert, over 70 per cent; Dubel, Tesler, von Ysendick, Kiewe, Reiche and Kaminer, between 50 and 60 per cent; Grisolle, Pradella, Tecklenberg, Neltner, Rosthorn, Fellner, between 10 and 30 per cent.

The wide divergence of these figures, running between 10 and 100 per cent shows clearly that they are either inaccurate, or based on clinical material which is not comparable. It seems that those who reported far advanced cases had a higher proportion of aggravations

[1] Am. Jour. Med. Sci., 1857, **34**, 87.
[2] Boston Med. Surg. Jour., 1897, **88**, 391.
[3] Jour. d. Practiciens, September 6, 1918, No. 36.
[4] Med. Ibera, 1921, **7**, 123.

and fatalities than those who dealt with incipient, or inactive cases of tuberculosis. More recent statistics, published by internists and specialists in hospitals for tuberculous patients, do not bear out the contention that pregnancy is as dangerous to all tuberculous women as the above figures, compiled mainly by gynecologists and obstetricians, would indicate. Thus Norris and Landis[1] report of 85 cases of pregnancy in tuberculous women observed at the Phipps' Institute at Philadelphia during a period of five years. They found that 85 per cent showed no change in their condition as a result of pregnancy. About 20 per cent of mild, quiescent, cases of pulmonary tuberculosis, and 70 per cent of advanced cases showed exacerbations during pregnancy or the puerperium. But taking a large number of cases in which pregnancy does not enter into consideration, we would expect to find about the same percentage of aggravations during the period of two years which cover pregnancy and lactation. Likewise, H. J. Forssner's[2] recently compiled statistics tend to show that the sinister influence of pregnancy on pulmonary tuberculosis is a dogma, a a tradition founded on unreliable information. He shows that extension of the lung disease may be synchronous with, but is not necessarily dependent on, pregnancy. He found that 881 patients observed during the period 1907–1912 were not better off than 133 who became pregnant. The exacerbation of the lung disease coincided with pregnancy in a certain number of cases, to be sure, but inquiry shows that this was merely coincidental. K. Schäffer,[3] in a study of the after histories of married women discharged from a sanatorium, arrived at about the same conclusion. Of 425 patients thus observed during a period of three to eighteen years, 136 had not become pregnant, and 189 had undergone from 1 to 7 pregnancies each since discharge. In 76.2 per cent the women were still fit for ordinary or light work despite the fact that they had been pregnant and gave birth to and raised children; only 18.4 per cent had died of tuberculosis, but only in 13.7 per cent was death due to the progress of the disease in connection with pregnancy. An examination of the after-histories showed that in the first stage 87 per cent, in the second, 83.3 per cent, and in the third, 63.6 per cent were still fit for work. Compared with 236 women who had left the sanatorium, and had not subsequently become pregnant, it appears that the former were better off. Only 53.9 per cent of the latter were fit for work and 38.4 per cent had died, as against 76.2 and 18.4 per cent respectively among those who had become pregnant. The author suggests that the non-pregnant fared worse because there were more far advanced cases among them. It may be added that, for this very reason, they may also have prevented pregnancy more assiduously than the others.

It has also been repeatedly stated that pregnancy seems to engender

[1] Ann. Rep. Phipps Inst., 1918, **14**, 1.
[2] Tubercle, 1920, **1**, 509.
[3] Ibid., p. 515.

tuberculosis in women "predisposed" to the disease. This is based on the statements of many patients to the effect that they had felt well until they conceived, and that during pregnancy, or soon after the puerperium, symptoms of phthisis made their appearance. Careful inquiry into these cases reveals the fact that many had been coughing for months or years before becoming pregnant, and inasmuch as 15 to 20 per cent of tuberculous individuals are likely to suffer aggravation in their disease during any year or two, the pregnant women appear not to be an exception. "Pregnancy and the puerperium, even when repeatedly occurring, are well and fairly well borne by a much larger proportion of tuberculous women than some pessimistic physicians claim," says Friedrich Kraus.[1] "In general it may be confidently stated that tuberculous disease with mild symptoms, which does not progress, the so-called 'quiescent' cases, which constitute about 90 per cent of all cases, suffer no aggravation of the disease as a result of pregnancy, labor, or the puerperium. The same is also true of about one-half of the number of active tuberculosis." Of course, it is different with patients in whom the disease is active and progressive, running fever, are emaciated and weakly, and especially those who have laryngeal and intestinal complications. This is the class that succumbs soon after childbirth. It is, however, noteworthy that it is extremely rare that a tuberculous woman should die with the fetus in utero. I have seen numerous pregnant women with far advanced and progressive lesions go through the long months of pregnancy, and die soon after the infant is expelled, but I have not seen one die before delivery. Moreover, there is no doubt that many tuberculous women feel much better, the symptoms of the disease abating to some extent, during pregnancy. Perhaps an explanation may be found in the circulatory changes occurring during pregnancy. It is well known that the mucous membranes of the respiratory tract are congested during that period, and this may retard the progress of the tuberculous process for the time being, as is seen to be the result of pulmonary congestion in cases of mitral stenosis (see p. 127).

Labor.—Many writers have suggested that the process of childbirth is predisposing to the evolution of phthisis and for this reason women who feel quite well during pregnancy develop active disease during, or soon after, the puerperium. Because of the straining at labor the intrathoracic pressure increases; in some cases to such an extent as to produce interstitial emphysema, in rare instances extending to the subcutaneous tissues of the neck. But this occurs only in extremely rare instances, and cannot be considered a strong predisposing factor. We have seen that quite often tubercle bacilli are found in the placenta (see p. 108). While separating from the uterus the sinuses are opened and tubercle bacilli may thus gain an entrance into the general circulation and cause a bacteremia. Also, as has been suggested by Hanau,

[1] Handbuch d. Tuberkulose, **3**, 280.

after labor the diaphragm descends and thus favors aspiration of tuberculous material from the upper parts of the lungs to the lower. But we have seen while discussing phthisiogenesis (see p. 151) that in tuberculosis metastasis does not, and there are indications that it cannot, take place in this manner. Nor can anemia, due to loss of blood during labor, be considered predisposing. The estimated amount of blood lost during normal labor is between 300 and 500 grams, and experience has shown that it soon regenerates through excessive work of the hematopoietic organs.

Experience has shown that only in cases of tuberculosis with active symptoms does progression downward follow pregnancy and labor. Those with latent, and many with quiescent lesions, pass through these processes unscathed. Obstetricians agree that difficult labor, involving manual or instrumental extraction of the fetus, does not occur more often than in non-tuberculous women. It is, indeed, often amazing to witness an emaciated, and evidently feeble woman pass through labor as easily as the average woman. Women who date back the onset of tuberculous disease to pregnancy, labor, and the puerperium are not much more numerous than might be expected when we bear in mind that a certain number of quiescent cases will reactivate within any given period. Perhaps the fact that during the child-bearing age, between fifteen and forty-five, the tuberculosis mortality is lower among women than among men of the same age period, shows best that the deletrious effects of pregnancy and labor have been exaggerated by some authors.

The Newborn Infant.—While congenital tuberculosis occurs, it is extremely rare (see p. 108); most of the infants born to tuberculous mothers are free from this disease at birth. To be sure, they are soon infected by the mothers, and the mortality among them is appalling, but even so, not an inconsiderable number have good chances of survival. Thus, of the infants born to tuberculous mothers observed during a five year period at the Phipps Institute in Philadelphia, Norris and Landis report that 14 were dead and 65 alive. They were found not unusually weak at birth, and the comparatively high death rate was due, not so much to weakness at birth, as to bottle feeding, and lack of care which an infant with a healthy mother enjoys. Schäffer reports on the fate of children born to tuberculous mothers under observation for three to eighteen years. Out of 356 pregnancies, 294 gave birth to live infants of whom 81.9 per cent had been, and still were, perfectly well. Of the remainder, 8.8 per cent were suffering from, or had died of, tuberculosis. Forssner reports his dispensary material among which he investigated the fate of 183 children who were healthy at birth and whose mothers suffered from pulmonary tuberculosis at the time of birth. During an observation period of twelve months to nine years, 64 per cent of these infants were alive and well, 13 per cent were tuberculous, and 23 per cent had died. But in only 13 per cent was death certified as due to tuberculosis. In another

group of cases, consisting of 283 infants, born to the same mothers, but before they began to attend tuberculosis dispensaries, it was found that in November, 1917, 73 per cent were alive and well, 19 per cent were tuberculous, and 8 per cent had died. Of 127 infants born to tuberculous mothers, 100 were cared for at home, 27 away from home. Of the "home" children, 67 per cent were alive and well, and 33 per cent had died of, or were suffering from, tuberculosis. Of the children cared for away from home, 89 per cent well, and only 11 per cent tuberculous.

It thus appears that, while the chances of contracting tuberculosis during the first few years of life are very great in infants born to tuberculous mothers, yet they are not all doomed. This is a very important point in prophylaxis, to which we shall return later on.

Extra-pulmonary Tuberculous Lesions.—It has been a time-honored custom of physicians to render a serious prognosis in cases of pulmonary tuberculosis when signs and symptoms of tuberculous lesions, past or present, of other organs are discovered. In "suspects," and in borderline cases, when there are noted scars indicating healed tuberculous lesion of the skin, glands, bones, or joints, or of active disease of these structures, the diagnosis of pulmonary tuberculosis is made without the positive proof which is exacted in persons in whom these stigmata are wanting. We have already shown that the prognosis in phthisis is no more serious in those with such scars than in those lacking them. But here we want to emphasize that there seems to be a distinct antagonism between tuberculosis of the lungs and that of other organs which is not appreciated to the extent it deserves; its diagnostic and prognostic significance cannot be overestimated.

Tuberculous Skin Lesions.—In sanatoriums and hospitals for tuberculous patients lupus, or any other tuberculous skin lesion, is extremely rare. In my hospital work it has been my experience that patients with tuberculous skin lesions only rarely have active lung lesions; when changes are discovered by physical exploration or roentgenography of the chest, the lesions are almost invariably latent, or healed; tubercle bacilli in the sputum are extremely rare in these cases. This fact has been established clinically and statistically by various authors, notably Jadassohn[1] Forchammer,[2] Lenglet,[3] Lewandowsky,[4] Bollog,[5] and many others. Our present views of immunity may explain this phenomenon: The lung lesion apparently endows the body with resistance against reinfection with the same virus. It is probably for this reason that, notwithstanding that tuberculous patients soil their hands, and other parts of their bodies, with sputum containing virulent tubercle bacilli, and at times sputum is deposited

[1] Die Tuberkulose d. Haut, 1914, p. 303.
[2] Arch. f. Dermatol. u. Syphilis, 1908, **92**, 3.
[3] La Pratique Dermatol., Paris, 1902, **3**, 239.
[4] Die Tuberkulose der Haut, Berlin, 1916, 64.
[5] Schweitz. med. Wchnschr., 1920, **1**, 939.

on open wounds of the skin, these organisms, as a rule, cannot gain a foothold and only rarely produce a lesion at the point of inoculation. Even when this does occur, as we see at times, there results a very benign process, akin to the "butcher's" or "pathologist's" warts; it remains local and does not implicate the regional lymphatic glands. In very rare instances we see real *tuberculosis verrucosa cutis* on the fingers of tuberculous patients. It appears that a similar antagonism exists between visceral and skin lesions in syphilis.

While tuberculous skin lesions are only rarely found in phthisical patients, it seems that the reverse does not hold to the same extent. Many patients have demonstrable tuberculous lesions in the lungs. But in most cases they are latent, or at most of a quiescent type, giving but little if any trouble. Jadassohn, John H. Stokes,[1] and many others, found that the lungs lesions in their tuberculous patients were benign. Lewandowsky suggests that while an active lung lesion may endow the body with a sufficiency of antibodies to prevent reinfection, a skin lesion cannot do it, because the skin does not offer a very good soil for the growth of tubercle bacilli (see p. 45). Another plausible explanation may be the following: Lupus begins mainly in children under twelve years of age. Among them pulmonary tuberculosis is very rare. When these children reach maturity, the dormant lung lesions may reactivate and produce symptoms. But Bollog found this not to be true among 400 patients with tuberculous skin lesions. However, it appears that, even so, the lung lesions are, as a rule, rather benign in individuals with lupus.

I have observed that in a few cases when the tuberculous skin lesions healed, pulmonary tuberculosis was not slow in making its appearance. The course of the disease in the lungs was acute and progressive. Recently a case of extensive lupus over the face, chest and arms was seen in which a very active lung disease developed.

There is one skin disease which is frequently associated with tuberculosis, especially miliary tuberculosis. I refer to *erythema nodosum*. However, it appears that only few investigators have found a tuberculous bacteriemia, or even tubercle bacilli in the skin lesions of erythema nodosum, while most others have failed to find them. From the extensive literature on the subject, which may be gleaned from the recent works of Lewandowsky,[2] Foerster,[3] and Stokes,[4] it appears that the relations of erythema nodosum to tuberculosis have not been firmly established. An attack of erythema nodosum, or the presence of tuberculides on the skin of infants and children, by no means implies that the child will succumb to acute miliary tuberculosis, and the writer has seen cases in adults running their course within a few weeks, without the subsequent development of pulmonary tuberculosis. Of

[1] Am. Jour. Med. Sci., 1919, **157**, 313.
[2] Die Tuberkulose der Haut, p. 212.
[3] Jour. Am. Med. Assn., 1914, **63**, 1266.
[4] Arch. Dermatol. and Syphilis, 1921, **3**, 29.

course, this does not exclude the possibility that erythema nodosum is caused by a tuberculous bacteremia.

Osseous and Articular Tuberculosis.—The vast majority of persons who present stigmata of glandular, osseous, and articular tuberculosis during childhood pass through life without developing pulmonary tuberculosis. On the other hand, it is exceedingly rare that a patient with an active lung lesion should develop a tuberculous lesion of a bone or joint. In patients with Pott's disease, or with ankylosed joints, pulmonary tuberculosis of an active and progressive type is rare. On this point many statistical investigations have been made, Wang[1] found among 2000 cases of clinical pulmonary tuberculosis only 20 with old cervical scars due to tuberculous lesions which had occurred during childhood. He observed that patients with such scars show a strong tendency to improve with the usual sanatorium treatment. Wallgren[2] observed the rarity of lung lesions in persons with old scars and the benign forms of pulmonary tuberculosis when it occurs in rare instances. Eliasberg[3] reports that tuberculosis limited to the lymphatic glands, bones and joints, during childhood tends to complete recovery. Nearly every patient with pulmonary tuberculosis admitted to the Montefiore Hospital during the past twelve years with either active or healed osseous, or articular tuberculosis was found to have healed, or quiescent, lung lesions, and when, in rare instances, the lung lesion was active, the progress of the disease was extremely chronic, tending to recovery. Demonstrable tubercle bacilli in the sputum are very rare.

An important exception is to be mentioned. These extra-thoracic tuberculous lesions appear to shield the lungs as long as the osseous or articular foci are not interfered with by surgical intervention. Soon after an operation the patient, very frequently, is attacked by acute and progressive tuberculosis in any of its forms, and succumbs. In others, after a long standing sinus has been healed by surgery, there appear symptoms of active pulmonary tuberculosis. This observation has been made by the writer repeatedly and for this reason a great deal of hesitation is indulged in before surgical intervention is advised in extra-thoracic tuberculosis. Most experienced surgeons also urge conservative treatment because the final results are much better.

Another striking confirmation of the immunity of the lung in extra-thoracic tuberculosis is often observed in families which are thoroughly tuberculized, and every child is attacked by some form of the disease. Those who develop glandular tuberculosis during childhood pass through life without lung lesions. W. Williams[4] puts it tersely in this fashion: "A very constant, if not invariable, fact I have observed in my fifty years' experience of tuberculosis is that those members of

[1] Jour. Am. Med. Assn., 1917, **68**, 1963.
[2] Upsala Läkareförennings, 1918, **23**, 1.
[3] Jahrb. f. Kinderheilk., 1919, **39**, 77.
[4] British Med. Jour., 1921, **1**, 158.

families with an inherited predisposition to the disease who suffer from cervical gland lesion leading to suppuration do not get pulmonary tuberculosis, or, indeed any other form of the malady; whereas several other members of the same families without gland lesions, and often more robust individuals, frequently develop consumption. All my cases have also shown signs that the apex of the corresponding lung had been affected but had recovered." Wallgren made the same observation. This has also been confirmed by observations during the World War. Several authors, notably Hayek,[1] found that soldiers showing no stigmata of past tuberculosis were more prone to develop pulmonary tuberculosis of a fulminating type, while those who had many signs of "predisposition" to the disease either escaped, or, when attacked, suffered from intensely chronic forms of pulmonary phthisis.

Genito-urinary Tuberculosis.—In patients with chronic pulmonary tuberculosis the kidneys are affected by the same process in about 15 per cent of cases. Among 128 autopsies of my cases we found that 22 showed tuberculous lesions of the kidneys; 8 were more or less severe caseous lesions and 14 miliary. In other words, the kidneys are most likely to be implicated in acute miliary tuberculosis which is so often a terminal phenomenon in chronic phthisis. It appears that as long as the lung lesion is active, excepting, of course, miliary cases, the lesion in the kidneys is in abeyance. On the other hand, most cases of so-called primary tuberculosis of the kidneys have very few symptoms and signs of active disease of the lungs. Here again operative interference is often followed by symptoms and signs of acute and progressive pulmonary tuberculosis.

It is well known that the epididymis and seminal vesicles are very prone to tuberculous changes, especially after slight injuries. In some, though not in as many as might be expected, these organs are found tuberculous in patients with long-standing and pronounced lung lesions. But in many others, the disease of the epididymis is apparently primary, inasmuch as no symptoms or signs of active lung disease can be discovered. It is important to remember that these patients do fairly well as regards their general health. For years the testicles may be swollen, tender, and annoying sinuses may be discharging. But they keep on gaining in weight, and acquire some of the stigmata of eunuchism: They become obese, listless, lazy, apathetic, and the secondary sexual characters become modified. In these patients cough, due to any cause, is at once attributed to pulmonary tuberculosis and proper treatment for this disease is instituted. But in most cases this is an error: Active and progressive pulmonary tuberculosis is rare among them.

It is important to emphasize that operative interference, which is only rarely successful in these cases, may be instrumental in reactivating the dormant lesions in the lungs which nearly all undoubtedly have.

[1] München. med. Wchnschr., 1919, **66**, 1316.

Another fact is that patients with tuberculosis of the male generative organs are more apt to succumb to tuberculous meningitis than those with lesions in other organs. M. Simmonds[1] found that fully one-third of 200 patients under his care died in this manner, while hardly 5 per cent of patients suffering from pulmonary tuberculosis succumb to meningeal complications. He also states that in 50 per cent of patients with tuberculous meningitis lesions in the generative organs may be found at the necropsies. This is especially true of tuberculosis of the seminal vesicles.

Diseases of the Respiratory Tract.—The Tonsils.—Hypertrophied tonsils, especially the lingual and the pharyngeal, are at present considered manifestations of the lymphatic diathesis. The extreme form of this constitution, the *status thymico-lymphaticus*, is comparatively rare, and may be left out of consideration. But hypertrophied tonsils and adenoids vegetations are very common, especially during childhood. In children with hyperplasia of the lymphoid ring in the throat, the cervical and at times the tracheo-bronchial glands are very often enlarged. These little patients cough, have fever at irregular intervals, and discharge mucopurulent material from the throat and nose; they are "subject to colds." Because of these symptoms, a diagnosis of tuberculosis is very often made in these lymphatic children; inasmuch as they present many of the stigmata of scrofula, they are at least classed among the strongly predisposed. Likewise, adults with hypertrophy of the tonsils, because of the well known symptoms they present, are often treated in the same manner.

We are begining to realize at present that lymphatism is distinctly antagonistic to active and progressive tuberculous disease of the lungs. Autopsies made by Bartel,[2] Symmers,[3] Emerson,[4] Ewing,[5] and others, have shown that active tuberculosis is rare in individuals of the lymphatic diathesis. Clinically we find that children, as well as adults with large tonsils, particularly with hyperplasia of the lingual and pharyngeal tonsils, only rarely develop pulmonary tuberculosis. Indeed, it appears that patients with active and progressive tuberculosis, as a rule, have small tonsils, and hardly ever adenoids. Moreover when in a case of mild tuberculous disease of the lungs the tonsils are removed surgically, the result is often that the lung lesion soon assumes an acute and progressive character; especially the larynx is liable to become implicated in the tuberculous process soon after the operation.

It is interesting in this connection that tuberculous patients only rarely suffer from the acute and epidemic infections of the nose and throat. Those with mild lung lesions are at times so affected, but

[1] Beitr. z. Klinik d. Tuberkul., 1915, **33**, 35.
[2] Status thymico-lymphaticus, Vienna, 1912; Pathogenese der Tuberkulose, Berlin, 1918.
[3] Am. Jour. Med. Sci., 1918, **156**, 40.
[4] Arch. Int. Med., 1918, **13**, 169.
[5] Jour. Am. Med. Assn., 1918, **71**, 1525.

those with acute or chronic progressive phthisis usually escape when other members of their families, or when the personnel of the sanatorium are stricken one after another. Even when affected, the acute coryza runs a mild course, hardly inconveniencing the patient. Clinical experience has taught that it is extremely rare that phthisis should begin with manifestations of an acute inflammatory process of the nose and throat. If we are satisfied that these were the first symptoms, we may safely exclude tuberculosis in doubtful cases.

Diseases of the Bronchi.—The acute and chronic forms of bronchitis have hardly anything to do with pulmonary tuberculosis etiologically. In its chronic forms, bronchitis is, as a rule, secondary to cardiac or renal disease, or to pulmonary emphysema. While it is at times convenient to maintain that a case which had been treated for bronchitis and the patient is indiscreet enough to have a pulmonary hemorrhage, or to begin to run fever, have nightsweats, etc., that the bronchitis has "turned into consumption," it is nevertheless a fact that such is hardly ever the case. Nor is there anything to the commonly accepted notion of tuberculosis having its origin in a "neglected cold." When the exciting cause of active tuberculous disease is exposure, the initial symptoms are those of pleurisy, or of tuberculosis with a localized lesion in the lung.

Chronic bronchitis, with cough for many years, with profuse expectoration, etc., hardly ever becomes tuberculous, and some authors have spoken of an antagonism between these two diseases. This is especially true of bronchiectasis. In a study of 40 cases of bronchiectasis Barth[1] found only in 1 the coexistence of tuberculosis, and he concluded that the two conditions are incompatible, a fact which Cruveilhier and Bamberger had observed before. The vast majority of cases under my care have not become tuberculous, though recently one who had been at the Montefiore Hospital for eighteen years with multiple bronchiectasis, with periodical profuse and threatening pulmonary hemorrhages, has suddenly, without any assignable cause, shown numerous tubercle bacilli in the sputum.

Chronic pulmonary tuberculosis is at times complicated by non-tuberculous bronchitis, and dilatations of the bronchi are found in many cases of fibroid phthisis that come to autopsy. Likewise, after attacks of influenza, tuberculous patients may remain with symptoms and signs of acute, or subacute, bronchitis which clears up sooner or later. In intensely chronic cases of phthisis with passive congestion of the lungs due to cardiac decompensation, chronic bronchitis is very frequent.

Asthma.—Many writers have stated that asthma and pulmonary tuberculosis are mutually exclusive, while others have maintained that the reverse is true. Considering asthma as a symptom, there is no doubt that it does occur in many cases of tuberculosis, especially

[1] In Herard, Cornil and Hanot's La Phtisie pulmonaire, Paris, 1888, p. 359.

the form known as fibroid phthisis. But carefully analyzing the difficulties in breathing met with in phthisis, it is clear that they are only rarely of the character seen in bronchial or essential asthma. The best appellation of the dyspnea in some cases of tuberculosis is that they are "pseudo-asthmatic."

It is noteworthy that when an asthmatic becomes tuberculous, which occurs only rarely, the paroxysmal attacks of dyspnea disappear, or are ameliorated. Overexertion is not, as a rule, a cause of dyspnea in bronchial asthma when there is no high-grade pulmonary emphysema with cardiac dilatation, as is attested by its nocturnal occurrence; it is in fibroid phthisis. On the other hand, afebrile tuberculous patients with nocturnal attacks of dyspnea, or with dyspnea on exertion, have a good prognosis as regards duration of life. While the pseudo-asthmatic attacks in phthisis are usually due to pulmonary fibrosis and cardiac dilatation, Bezançon and de Jong[1] have recently shown that they may also be caused by anaphylaxis, as is the case in many forms of bronchial asthma. However, it appears that juvenile asthma has nothing to do with tuberculosis. The crystals, spirals, and eosinophile cells are almost invariably found in asthmatic sputum; they are lacking in tuberculous sputum, excepting eosinophiles, which are now and then seen is phthisis.

Pulmonary Emphysema.—Compensatory emphysema of the unaffected portions of the lungs occurs in nearly all cases of chronic tuberculosis in which there is extensive destruction of the parenchyma. But this is of a vicarious nature. On the other hand, persons with atrophic emphysema only rarely become tuberculous, as has been pointed out long ago by Rokitansky. The following has been offered as an explanation as to why an atrophic lung is a poor soil for tubercle bacilli: Tuberculosis occurs mainly in individuals between eighteen and forty-five years of age, while pulmonary emphysema is a disease of persons over forty. And it is among persons of advanced age that we actually find the two diseases combined. However, the emphysematous form of fibroid phthisis, as well as phthisis in the aged, run an intensely chronic course, without fever, and with slight cough and expectoration; but dyspnea and cyanosis are very common in the last stages. The diagnosis is at times very difficult because the large emphysematous lung screens the tuberculous lesion to an extent as to render physical examination, and often roentgenography, of little value in attempts at localization of the lesion. In many cases only positive sputum decides, but in a large proportion it is negative.

Pneumonia.—Tuberculous patients only rarely develop genuine lobar pneumonia, and during epidemics of influenza the characteristic bronchopneumonia occurs only in comparatively few of those who have active tuberculous lung lesions. Now and then we meet with a case of true lobar pneumonia in a tuberculous patient. The symp-

[1] Presse Méd., 1920, p. 885,

toms are about the same as in others, with sudden onset, chill, fever, pain in the chest, etc. The character of the expectoration often changes into the rusty, viscid material characteristic of pneumonia. It is noteworthy that the course does not materially differ from the usual, the crisis occurring in seven to twelve days (see Fig. 113), and when termination is by lysis, the cause is usually to be sought in the activity of the tuberculous lesion, or in complicating empyema.

It has been our experience that in most instances the lower lobe was affected and physical signs of consolidation are easily made out; rarely apical involvement is observed, and then differentiation from caseous pneumonia is extremely difficult. Bacteriological and serological tests are not of great value because both organisms, tubercle bacilli and pneumococci, may be found in the sputum in either case. The most experienced clinician may be deceived, diagnosing an extension of the tuberculous process in the lungs, or caseous pneumonia, and render an unfavorable prognosis to be astonished when, within a week or ten days, the temperature drops by crisis, and the patient goes on with his phthisis.

FIG. 113.—Temperature curve in a case of lobar pneumonia complicating pulmonary tuberculosis.

Bronchopneumonia occurring in tuberculous patients is almost invariably of a caseous nature and should be considered an ill omen. An exception is influenzal bronchopneumonia which, as a rule, runs a less severe course in the tuberculous than in those who were healthy before the onset of the epidemic disease. But tuberculous patients are often left with signs and symptoms indicating involvement of new areas of lung tissue. However, these newly involved areas, in many cases, clear up sooner or later, while the tuberculous lesion keeps on its course as if no complication had occurred.

On the other hand, the prognosis of lobar pneumonia in tuberculous patients is rather favorable. Most patients recover and no trace is left behind, the phthisical process pursuing its course as might be expected. This was already noted by Walshe who said: "If lungs, already tuberculized, become acutely inflamed, convalescence from the pneumonia often takes place as rapidly, as if the lungs had previously been sound; and no increase in activity of the tuberculous disease

necessarily follows." Grisolle, Wilson Fox, and many others, also found that pneumonia exerts little or no influence on the course of phthisis. Some writers have even maintained that the pneumonia process has a rather salutary effect on the tuberculous lesion. Thus, W. J. Pettit[1] arrives at the conclusion that intercurrent pneumonia during the course of phthisis is not unfavorable, but appears to have been an important factor in the production of a cure. He compares it with the effects of erysipelas which has repeatedly been observed to cure chronic indolent tuberculous ulcers, and he is inclined to assume that a similar reaction occurs in the lungs. Ernst v. Czyhlar[2] has made similar observations.

There remains yet to mention the fact that lobar pneumonia is hardly every followed by pulmonary tuberculosis. The residual lung lesions after lobar and bronchopneumonia are abscess and, rarely gangrene of the lung, chronic intersititial pneumonia, with or without the formation of bronchiectasis, or localized chronic bronchitis. The last is especially apt to follow influenzal pneumonia. In most cases which are considered as "unresolved pneumonia" it is found that the lesion is a localized or encapsulated empyema, but tuberculosis is exceedingly rare. To be sure, many tuberculous patients give a history of pneumonia at the beginning of their malady. But an investigation, whenever possible, usually reveals that an acute onset of pulmonary tuberculosis was mistaken for pneumonia.

Diseases of the Circulatory Organs.—The organs of circulation are affected by tuberculosis in two ways, *viz.*, by the toxic effects, and mechanically. The toxemia of phthisis is effective in producing hypotension, tachycardia, and instability of the pulse-rate. In cases in which the toxic action is prolonged and intense, myocarditis is the rule; in the milder cases cardiac neuroses, and instability of the vasomotor functions are noted. These effects may be seen in early cases. They may disappear with the decline or disappearance of the fever.

With the advance of the disease, when destruction of pulmonary tissues has gone quite far, mechanical effects manifest themselves in the heart and bloodvessels. This is especially seen in fibroid phthisis in which the cardiac symptoms may prove more annoying than those resulting from the tuberculous process *per se*, and the toxemia, which is lacking in many cases. Because of the obliteration of many pulmonary bloodvessels, the heart finds difficulty in propelling sufficient blood to the systemic circulation. The result is enlargement and dilatation of the right heart, and stasis in the veins. Many authors notably Regnault,[3] Bohland,[4] Sokolowski,[5] and many others, speak of hypertrophy and dilatation of the right heart in chronic cases of

[1] Jour. Am. Med. Assn., 1912, **58**, 852.
[2] Beitr. z. Klin. d. Tuberkulose, 1911, **21**, 33.
[3] Le coeur chez les tuberculeux, Paris, 1899.
[4] Handbuch d. Tuberkulose, **4**, p. 5.
[5] Arch. f. klin. Med., 1885, **27**, 443.

tuberculosis. But a careful study by modern methods made by Boas and Mann[1] of material at the Montefiore Hospital showed that with the aid of electrocardiography right ventricular predominance is not found more frequently in association with fibroid phthisis, or with pleural adhesions, than with other types of pulmonary tuberculosis. In fact, autopsy experience shows that a large heart is very rare in fatal cases of pulmonary tuberculosis.

Clinically it is, at times, difficult to determine with exactitude whether the dyspnea, acrocyanosis, edema, enlargement of the visceral organs, notably the liver, are of cardiac or pulmonary origin. One of the most convenient ways to solve this problem is to put the patient to bed at perfect rest for a week or two. If this is effective in reducing the rate of the pulse, ameliorate the dyspnea, etc., it is clear that the heart is, in a great measure, responsible. However, in toxic myocarditis rest, instrumental in reducing the toxemia, may also improve the quality of the circulation.

In chronic cases other mechanical impediments to the circulation are frequently observed. Partial or complete obliteration of the pleural cavity, and pleuro-pericardial adhesions, resulting in cardiac displacements, as has been described elsewhere,[2] are effective in producing dyspnea, cyanosis, etc. Adhesive pericarditis is very common, especially in left-sided lesions. In many cases the tuberculous lesion in the lung heals more or less, or remains quiescent for years, but the patients remain short-winded, with acrocyanosis, etc., and a diagnosis of "heart disease" is made.

In these cases murmurs are often heard over the region of the cardiac apex. Commonly the murmur is systolic in time and may be transmitted to the axilla. In rare instances I have heard a presystolic murmur over the mitral area, but at the necropsy no changes were found in the valves to account for it. It is these cases that are often brought forward as proof that the coexistence of mitral stenosis with pulmonary tuberculosis is not unusual. Retraction of the left lung which contains a large cavity exposes the heart, and a thrill may be felt over the cardiac apex, but even this should not invariably be taken as proof that there is an obstruction to the flow of blood through the mitral valve. These murmurs are clearly the result of cardiac displacements and pleuro-pericardial adhesions.

The effects of pulmonary tuberculosis on the circulatory organs are also seen in the terminal stages of the disease when myocarditis of toxic origin, and also tuberculous implication of the adrenals, result in heart failure, extremely low blood pressure, terminal edema of the extremities, edema of the lungs, etc. Some of these patients, apparently holding their own, die suddenly owing to heart failure.

Effects of Heart Disease on Pulmonary Tuberculosis.—Many authors have stated that congenital hypoplasia of the heart muscle is a pre-

[1] Arch. Int. Med., 1921, **28**, 62.
[2] Fishberg: Arch. Int. Med., 1914, **13**, 656.

requisite, or a predisposing factor, for tuberculosis (see p 127). Clinical, roentgenographic, and autopsy confirmation is not lacking. It appears, however, that in the vast majority of cases of early phthisis the heart is normal in size, and that only with the progress of the tuberculous disease it participates in the wasting process of the organs of the body, especially the muscles. In other words, the small heart seen in phthisical patients is to be considered an expression of the general cachexia of phthisis, a phenomenon observed in other wasting diseases, notably cancer.

Some eighty years ago Rokitansky asserted that disease of the heart and bloodvessels, producing passive congestion of the lungs, are preventive of phthisis. Further clinical observation and autopsy records embracing thousands of cases have confirmed the opinion of this pioneer pathologist. It appears that individuals suffering from mitral stenosis are only rarely affected with active and progressive tuberculosis of the lungs.

This antagonism, as far as we know at present, refers only to tuberculous disease. Infection with tubercle bacilli occurs in cardiacs practically as frequently as in others. But, owing to the congestion and plethora of the lesser circulation produced by the stenotic cardiac valves, active tuberculosis is prevented. On the other hand, in congenital heart disease, pulmonary stenosis, tuberculosis is the cause of death in nearly all who have survived the first decenium. Here the condition is just the opposite of that in mitral stenosis. The blood pressure in the pulmonary artery and lungs is low, and there is a distinct anemia of the lung tissues.

Persons suffering from disease of the aortic valve at times develop pulmonary tuberculosis, but the lung lesion, as a rule, pursues a very benign course. This observation has been made by the writer repeatedly; no acutely progressive case of phthisis has been seen in a patient with aortic valve disease, especially when this is accompanied by aortitis. Perhaps the most plausible reason that can be assigned is that aortic disease is often due to tertiary syphilis, or atherosclerosis, both of which are accompanied by sclerotic changes in various visceral organs, including the lungs. On the other hand, in cases of aneurism of the aorta tuberculous lesions at times develop in the area of lung tissue contiguous to the blood tumor. Here we deal with tuberculosis favored by compression of the pulmonary parenchyma, which is at times also observed in intrathoracic malignant neoplasms. They are of little interest clinically, though the pathologist may find suggestive hints for further study of these cases.

Infectious Diseases.—The Exanthemata.—Among the acute endemic diseases of childhood, scarlet fever is only rarely followed by tuberculosis, and some writers have maintained that there is a distinct antagonism between these two maladies (Rilliet and Barthez). The acute sequels of scarlet fever are mainly suppurating glands, nephritis, and arthritis. These, as will be shown later on, are distinctly antago-

nistic to the development of tuberculosis. The writer has not observed any reactivation of tracheo-bronchial adenopathy after scarlet fever, as is the case with measles. The same is true of osseous and articular tuberculous lesions.

During epidemics of measles very few tuberculous children escape, and after the acute exanthema has passed many show symptoms and signs of reactivation of the tuberculous process. Intrathoracic glands, which may have given no, or only slight, trouble, become actively diseased, suppurate and involve neighboring chains; even tuberculous lesions in bones and joints have been observed to reactivate after an attack of measles. A large proportion of cases of bronchopneumonia following measles is tuberculous, and is probably due to rupture of suppurating intrathoracic glands and flooding the lungs with tuberculous pus. However, this seems to be mainly the case with children; in adults, measles appears to have no such effect. The extensive epidemics of measles among our troops during the World War have not increased the morbidity and mortality from pulmonary tuberculosis. At any rate, there seems to be no evidence to this effect.

With smallpox we have had no experience, but writers of former generations stated that there seemed to exist some antagonism between it and tuberculosis. Antivaccination agitators have maintained that vaccinia predisposes to the development of phthisis. But the fact that the mortality from tuberculosis has declined in all civilized countries in which vaccination has been general speaks against this view. Some have mentioned the possibility of transmitting tuberculosis by vaccination. But there is no authentic case on record of this ever having occurred.

Whooping Cough.—Latent tuberculosis is often reactivated by pertussis and, in children, tuberculous bronchopneumonia is one of the most dreaded sequels of this disease. The combination of pertussis and tuberculosis in adults has been observed by the writer in several instances. In all the tuberculous process was aggravated, and in one it seemed as if the whooping cough was responsible for the fatal issue.

Influenza.—The recent pandemics of influenza have shown that this disease hardly has any influence on the origin, course, and termination of pulmonary tuberculosis. The fact that during the three years following the epidemics the mortality from tuberculosis has declined, favors this view. Indeed, the death rates in sanatoriums and hospitals for consumptives which were visited by epidemic influenza have not shown any rise during the past three years (see p. 128).

It appears from available evidence, which is quite ample in this case, that tuberculous patients are no more liable to suffer from the effects of influenza than non-tuberculous. Many writers are under the impression that individuals with active tuberculous disease manifest a certain degree of immunity against influenza; that proportionately fewer are attacked and, when affected, are less likely to fall prey to complicating bronchopneumonia than others. The low rates of mor-

tality and morbidity, during the epidemics, of tuberculous patients compared with the administrative staffs of these institutions, has raised the question why the weaker stand more chance of survival during epidemics of influenza than the healthy and vigorous.[1]

The symptoms and course of influenza does not materially differ from those observed among others. In but few has an extension of the tuberculous process in the lungs been observed. The liability to pneumonic complications appeared much less than in previously healthy individuals. After four to six days of fever, weakness, in some instances hemoptysis, etc., the symptoms abated, and the original disease, tuberculosis, pursued its course as if no complication had occurred. The fever chart (Fig. 114) is characteristic of this class of cases, which were in the majority. As far as physical signs indicated, no changes were found in the tuberculous lesions in most cases.

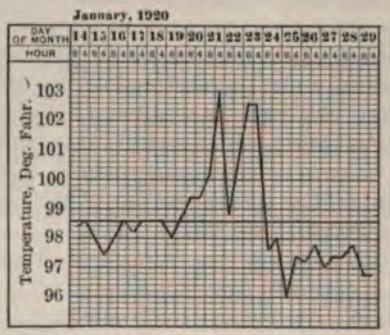

FIG. 114.—Uncomplicated influenza in a case of inactive tuberculosis.

In a small minority of cases, irrespective of the stage of the tuberculous disease, though there seems to be evidence to the effect that incipient and quiescent cases were more liable to this complication, bronchopneumonia developed. In afebrile cases of tuberculosis the temperature rose, reaching 103° to 105° F., rarely higher (Fig. 115). In the majority of instances the onset was gradual, though in a few it was sudden and preceded by a chill. Dyspnea and cyanosis were prominent symptoms in most cases, and weakness, at times prostration, was pronounced. At this period, the third or fourth day, physical signs of bronchopneumonia may be made out over one base, or both—diminished breath sounds, moist rales, at times bronchial breathing, and dulness. It is noteworthy that only rarely have old tuberculous lesions given indications of reactivation; in most cases the physical signs of the tuberculous lesion remained unaltered.[2]

In most instances the fever kept on for ten days to two weeks; in others it lasted for several weeks (Fig. 116). Deferred defervescence

[1] See Fishberg: Am. Rev. of Tubercul., 1919, **3**, 532.
[2] See Fishberg and Boas: Am. Jour. Med. Sci., 1920, **160**, 214.

was, however, rare. In patients with advanced and progressive tuberculosis the inevitable was simply hastened by the complicating influenza; but this was not the rule in our cases. Many far-advanced cases passed through uneventfully, and the tuberculous process then pursued its

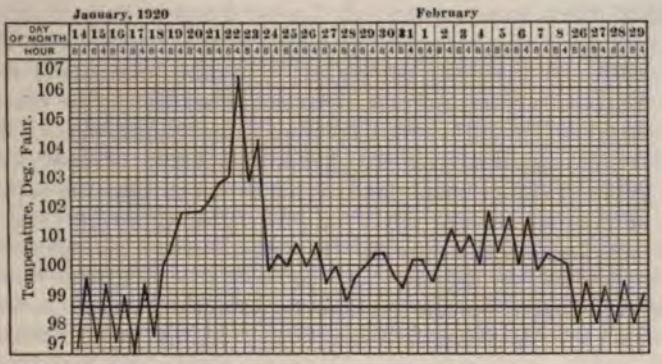

FIG. 115.—Influenzal bronchopneumonia. Temperature curve uninfluenced by previous fever.

course. On the whole, it appears that the outlook for recovery was much brighter in moderately advanced than in incipient cases. It was surprising that in the majority of cases no after effects were noted. In some instances the residual bronchial catarrh remained, but even this disappeared in time. In non-tuberculous, influenza leaves, at times, signs of localized pathological processes in the bronchi, lungs,

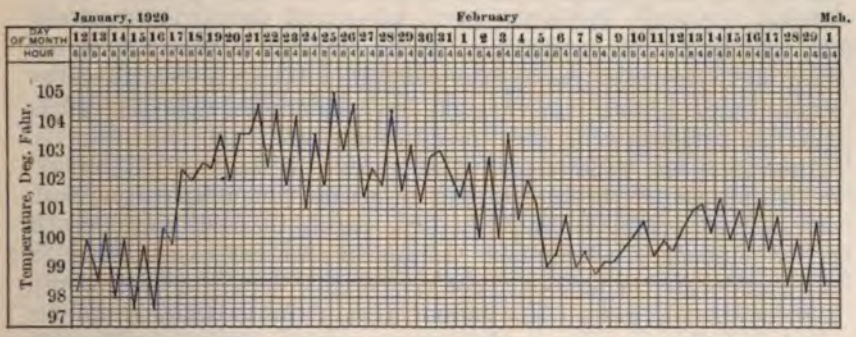

FIG. 116.—Influenzal bronchopneumonia. Slow return of fever to the pre-influenzal type.

and throat, causing chronic cough, expectoration, etc. These are often mistaken, and treated for tuberculosis. It is important that they should be differentiated. Details about the diagnosis of these lesions are given elsewhere (see p. 542).

Typhoid Fever.—That typhoid fever has not been shown to be a predisposing factor to tuberculosis has already been stated (see p. 129). Recently the problem of the coexistence of the two diseases has come in for discussion because of the problem of reactivation of dormant tuberculous lesions by antityphoid inoculation. Formerly several French authors, notably Andral, Louis, Forget, Pidoux, etc., considered typhoid altogether antagonistic to tuberculosis and that the two diseases are mutually exclusive. Villemin pointed out the similarity of onset in many cases, and insisted that this is the reason why some consider typhoid a reactivator of latent tuberculosis; he concluded that, in a general way, they are antagonistic. Recent experience with typhoid in tuberculous patients have shown that the superadded infection has no serious effect on the tuberculous process, or the symptomatology of the disease. It was observed that patients with inactive tuberculous lesions may have typhoid fever without any detrimental effects on the pulmonary lesion. During epidemics of typhoid at the Trudeau, and the West Virginia State Sanatoriums, Brown[1] and his co-workers, and Clovis and Mills,[2] did not observe any aggravations of the pulmonary process. Most patients recovered. It is noteworthy in this connection that several authors report cases of acute miliary tuberculosis with typhoid bacteremia. But in these cases, described by Busse and Bloomfield, it cannot be justly stated that the typhoid bacteremia had a deleterious influence on the tuberculous process which was acute, and would have proved fatal without the superadded typhoid infection.

Syphilis.—Tuberculosis and syphilis being very widespread and lasting for many years in the affected patients, it is to be expected that the two diseases should often be found in the same person. Some authors, especially in France, have maintained that syphilis reduces the natural resistance against tuberculosis, and is thus one of the most important predisposing factors in phthisiogenesis. On the other hand, beginning with Hunter, many have maintained that there exists a certain antagonism between these two diseases.

Syphilis in the Tuberculous.—The association may be encountered in two forms: A tuberculous patient acquires syphilis; or one with old, tertiary syphilitic manifestations, begins to show symptoms of active pulmonary tuberculosis. Empirically we have learned that in the former case the outlook is not very bright, though not necessarily hopeless, in most cases. The patient, already troubled with symptoms of phthisis, loses courage and as soon as the diagnosis of chancre is made, he becomes more or less despondent. If the lung lesion is very active, especially if it has been running an acute and progressive course, antisyphilitic treatment often aggravates the outlook, and the prognosis is grave. In fact in several cases the writer was convinced that the patients perished as a result of the combination of the two diseases.

[1] Am. Rev. Tubercul., 1919, 717.
[2] Jour. Am. Med. Assn., 1920, **74**, 297.

It must also be mentioned that in cases in which the lesions of both diseases—the tuberculous lung lesion, and the secondary manifestations of syphilis—are active and manifest tendencies to progression, treatment is beset with painful obstacles. This is especially true of the specific treatment of syphilis. Both mercury and arsphenamine at times aggravate the acute lung lesion, and may induce copious pulmonary hemorrhages.

In intensely chronic tuberculous cases, and in those in whom the disease is rather quiescent, things are different. In them, antisyphilitic treatment may be administered with benefit. However, even here the lung lesion must be watched for reactions when arsphenamine, mercury, and potassium iodide are administered. At the least rise of the temperature, or attack of hemoptysis, the specific treatment is to be discontinued, or restrained.

Tuberculosis in Patients with Tertiary Syphilis.—The contrary appears to be true of patients with old syphilitic lesions who develop symptoms of pulmonary tuberculosis. In them the prognosis of the tuberculous lesions is, as a rule, more favorable than in others. Many writers speak of an antagonism between syphilis and tuberculosis; at any rate, it has not been proved that syphilitic subjects are more apt to develop phthisis than others. On this point there seems to be an agreement among recent authors, such as S. West, Percy Kidd, D'Arcy Power, Sir Dyce Duckworth, and many others. This point is well summarized by Jonathan Hutchinson,[1] who takes the broad view of the subject and concludes that "a tendency to tuberculosis may be modified by the introduction of syphilitic poison, not that the latter poison may predispose to, or encourage the development of tuberculosis."

The cardinal pathological changes in tertiary syphilis are fibrosis of the various visceral organs and bloodvessels, and fibrosis is Nature's mode of curing tuberculosis. Though it appears that the lung is one of the few organs in the body which escape the action of the syphilitic virus very often, still, when tuberculous lesions do occur in old syphilitics, they are very often fibrous in character. Several French authors, notably Sergent,[2] insist that in all cases of fibroid phthisis a careful search should be made for stigmata of tertiary syphilis. He maintains that inequality of the pupils, absent or sluggish reaction of the pupils to light, and particularly leukoplakia of the buccal mucous membrane, etc., are very frequently found in cases of fibroid phthisis, and also in others in whom old and chronic bronchitis, pulmonary emphysema, etc., are diagnosed.

My personal experience has not been in agreement with that of Sergent; a positive Wassermann reaction had only rarely been obtained among the numerous cases of fibroid phthisis in the Montefiore Hospital, indicating that the vast majority of cases of fibroid phthisis have no syphilitic substratum. However, most cases of pulmonary tuber-

[1] D'Arcy Power's System of Syphilis, London, 1912, **2**, 12.
[2] Études cliniques sur la tuberculose, Paris, 1919, p. 247.

culosis in patients with tertiary syphilis have been found to run a benign and sluggish course: The patients cough, expectorate, have pulmonary hemorrhages for many years, but are, as a rule, afebrile. The most annoying symptom appears to be dyspnea, which is only rarely lacking. It is noteworthy that emaciation is not a common symptom; the majority of the patients are more or less obese. Because demonstrable tubercle bacilli are rare in the sputum, many of these patients are treated for years for bronchitis, asthma, pulmonary emphysema, etc. In many instances there are syphilitic lesions which skilful and experienced laryngologists find difficult to differentiate from tuberculous laryngitis and only the therapeutic test clears up. But even the pulmonary lesion is not easily diagnosed. The "hybridity of the lesions," as Sergent refers to them, is often perplexing to the clinician. The results of the Wassermann test are not always decisive for obvious reasons. One of the most important features of these cases is the high blood-pressure. In all cases of tuberculosis with vascular hypertension, a reason should be sought, and it will be found that, in addition to the specific lung lesion, there is either a cardiac, renal or gouty substratum, or, more commonly, tertiary syphilis.

The most important feature of the association of pulmonary tuberculosis with tertiary syphilis is that the prognosis is very good, and that antisyphilitic treatment, arsphenamine, mercury, and the iodides, render excellent therapeutic service in relieving the symptoms of both diseases. It is probably for this reason that many writers, in this country B. L. Wright, have reported success with mercury in the treatment of pulmonary tuberculosis. More recently arsphenamine has been urged in some cases. A French author, G. Milian,[1] speaks of *heterotherapy*, the treatment of a disease with the specific medication for another disease, in this connection.

It is curious that several writers, having observed the good prognosis of tuberculosis in syphilitic subjects, have suggested that inoculation with the syphilitic virus might be advisable for therapeutic purposes.[2] This is mentioned merely as another aspect of the many curious "cures" that have been suggested for tuberculosis.

Mixed Infection.—Soon after the discovery of the tubercle bacillus many authors, finding pyogenic microörganisms in the secretions and excretions of tuberculous patients, maintained that most of the symptoms of phthisis are due to mixed infections. Especially the fever, nightsweats, amyloid changes in the visceral organs, etc., were attributed to superinfection with streptococci, staphylococci, pneumococci, etc., which are found in the sputum, the contents of cavities, as well as their walls, which are often covered with pyogenic membranes. This view, held by Cornet, Petruschky, Maragliano, and many others, is well expressed by Victor C. Vaughan: "Unaided, the tubercle

[1] Paris Médicale, 1920, **10**, 337.

[2] See G. Z. Petresco: Tuberculose et Syphilis, 1903; Quoted from Ztschr. f. Tuberkulose, 1904, **6**, 468.

bacillus seldom kills, but the microbic tissues caused by its growth form a suitable medium for the lodgment and growth of other bacteria, and tuberculosis usually terminates as the result of infection. So long as the infection is unmixed, the progress of the disease is slow." That this view is not exactly in agreement with known facts is seen in acute general miliary tuberculosis, which is invariably fatal, and it is here that only the specific germ is found. On the other hand, many advanced cases of phthisis, with large cavities, the sputum containing large numbers of pyogenic microörganisms in addition to the tubercle bacilli, have no fever, anorexia, nightsweats, emaciation, etc.

The term mixed infection has not always been properly applied by many authors. Secondary, or superimposed, but transient infections, occurring during the course of phthisis, such as influenza, pneumonia, etc., are no more mixed infections than is diphtheria, gonorrhea, or malaria. A patient suffers from mixed infection when, in addition to the tubercle bacilli, other pathogenic microörganisms have entered the tissues of the body and are influencing the course of the disease permanently, or for a long time. Some authors, in this country Roswell T. Pettit,[1] have maintained that this is often the case in phthisis. Pettit isolated pneumococci and streptococci from the blood of more than one-third of cases of tuberculosis, and he concludes that "not only are true secondary invading organisms of frequent occurrence in pulmonary tuberculosis, but further, that in many instances these organisms, entering the blood stream, constitute a complication of extensive pathological significance."

Other authors, with a view of proving the importance of mixed infections with pyogenic microörganisms, have pointed to the blood count in this disease. But we have shown that a real leukocytosis is rare in phthisis, and particularly in the acute miliary form of the disease (see p. 444). The increase in the number of white blood cells to 12,000 or 15,000 does not constitute leukocytosis due to septic infection.

It must be emphasized in this connection that when pyogenic microörganisms are found the sputum, or in the contents of cavities, it does not necessarily prove that they are responsible for the symptoms. In the first place, they may not have come from the lungs but altogether from the upper respiratory passages and the mouth. To be sure, various methods have been devised to wash the sputum which has been carefully collected, but according to painstaking bacteriologists they have proved unsatisfactory. Radcliffe,[2] in fact arrives at the conclusion that "when a true secondary infection is present, it is impossible to remove the causative microbe from the sputum by washing, just as it is impossible to remove the tubercle bacillus."

The weight of current opinion is against the contention that the symptoms of tuberculosis are in a great measure due to superimposed

[1] Jour. Infect. Dis., 1911, **9**, 237.
[2] Ztschr. f. Tuberkul., 1913, **21**, 258.

infections. To be sure, in more than 50 per cent of cases of chronic phthisis pyogenic germs are found in the sputum, and also in the walls of pulmonary cavities. But it appears that they exist merely as saprophytes. Some authors have, in fact, referred to this form of superinfection as *passive mixed infection*, giving no symptoms. At any rate, clinically it cannot be identified, because the symptoms caused by the tubercle bacilli cannot be differentiated from those produced by the superadded microbic invasion. Acute exacerbations during the course of phthisis are, at times, undoubtedly due to these mixed infections, but the hectic fever, which bears great similarity to pyogenic septic fever, amyloidosis, etc., have been proved to be due to the tubercle bacilli, and the absorption of toxins from the decayed tuberculous tissues in the lungs.

A non-tuberculous bacteremia is extremely rare in phthisis. Investigations by Sorgo,[1] Radcliffe, Reiche,[2] Jochmann,[3] Brauer and Peters,[4] and many others, have shown that the blood is sterile in nearly all cases of chronic and progressive tuberculosis, excepting, of course, that in some cases tubercle bacilli are found (see p. 284). Streptococci and staphylococci may be found in very rare instances in the blood of tuberculous patients, but this is usually a terminal phenomenon, occurring a few days before death when the defensive forces of the body have been paralyzed.

Constitutional Diseases.—The group of diseases classed as "constitutional" offers some very good examples of the effects of other pathological processes on the origin and course of pulmonary tuberculosis. Among these the most important are those manifesting themselves as chronic rheumatism, gout, migraine, neuralgia, myalgia, obesity, asthma, stone in the kidney and gall-bladder, premature atherosclerosis, diabetes, etc. In short, the group called by the French "arthritism" and in English and American literature "lithemia," or the arthritic diathesis. It appears that, with the exception of diabetes, persons afflicted with any of these diseases are comparatively immune to progressive pulmonary tuberculosis. When tuberculosis does occur, it runs a mild, benign and sluggish course.

This was already noted by Morton, who, in his *Phthisiologia*, published in 1689, said "arthritic phthisis is an asthmatic form of the disease, noteworthy more for the viscosity of the bronchial secretions than by its obstinate cough."

Rheumatism.—Tuberculous rheumatism was first described by Chamorro in 1888, Grocco in 1892, and finally by Poncet[5] in 1897. According to these authors tubercle bacilli or their toxins, are frequently

[1] Wien. klin Wchnschr., 1904, **17**, 725; Ztschr. f. klin. Med., 1907, **61**, 250.
[2] Med. Klin., 1909, **5**, 1962.
[3] Deutsch. Arch. f. klin. Med., 1905, **83**, 558; Lehrbuch d. Infektionskrankheiten, Berlin, 1914, p. 154.
[4] Handb. d. Tuberkulose, 1919, **3**, 148.
[5] The literature on this subject may be gleaned from Poncet and Leriche's Le Rheumatisme tuberculeux, Paris, 1909.

the cause of acute and chronic mono- and polyarthritis, and various arthralgias, at times running a febrile course indistinguishable from acute articular rheumatism and subacute and chronic pains in the joints, including the spinal column which may or may not end in ankylosis. In the chronic cases the general condition of the patient remains good, and cardiac complications are rare, while the joint symptoms are pronounced. Whether the arthritic symptoms in these cases are really caused by the tubercle bacillus or its toxin, is a disputed problem and will not be entered into here.

One clinical fact is, however, important for those who care for tuberculous patients. It appears that patients with rheumatic troubles involving one or more joints, presenting any of the clinical syndromes of chronic or subacute rheumatism, only rarely suffer from active and progressive pulmonary tuberculosis. There are admitted to my service at the Montefiore Hospital annually several cases of arthritis deformans, chronic rheumatism, spondylitis, etc., with some symptoms and signs of localized lesions in the lungs or pleura. But in nearly all no symptoms of active pulmonary tuberculosis can be observed. In the extremely rare instances in which there are demonstrable tubercle bacilli in the sputum, thus showing that the lung changes are undoubtedly tuberculous, the patients are afebrile and usually well nourished, indicating that the process in the lung is in abeyance.

The same is true of gout. Wunderlich found among 108 gouty persons that not one presented symptoms of tuberculosis; Cotton states that among 1000 tuberculous patients only 6 showed symptoms of gout.[1] At the Montefiore Hospital experience has taught us that wherever we meet a case of gout, or chronic rheumatism admitted to the tuberculosis division with a diagnosis of tuberculosis, there are either no symptoms of active lung disease to be discovered after a careful and prolonged study of the case, or when the lesion is tuberculous, it is of the sclerotic form, hardly giving any symptoms. Well-nourished consumptives, the "fat consumptives" mentioned elsewhere (see p. 606), are mainly found among arthritic subjects, or perhaps of arthritic stock, and also among those who were scrofulous during early childhood. Some of these patients have hemoptyses, at times copious hemorrhages occur, but a fatal issue is extremely rare; some even feel better after the bleeding. Pidoux,[2] Sokolowski,[3] Lemoine,[4] and others pointed out that the arthritics supply the main contingent of curable case of phthisis, and those who, despite tuberculosis, reach an advanced age.

English authors, whose experience with gout has been extensive, confirm these observations. J. E. Pollock believes that "gout, like

[1] In Herard's article on tuberculosis in Herard, Cornil and Hanot's La Phtisie Pulmonaire, Paris, 1888, p. 367, numerous references are made to the literature on this subject.

[2] Études générales et pratique sur la phtisie, Paris, 1873.

[3] Deut. Arch. f. klin. Med., 1890, **47**, 558.

[4] Semaine Médicale, 1900, **20**, 103.

rheumatism, when the specific attack of the disease is developed in a case of tubercle, retards the latter." Sir Dyce Duckworth also considers gout, or the gouty diathesis, as antagonistic to phthisis. F. Parkes Weber[1] suggests that the resistance of gouty person toward tuberculosis is partly due to the meat food (butcher's meat, eggs, and all animal protein foods) which most persons with acquired goutiness have been accustomed to indulge in freely during most of their lives. "Great meat eaters, if not alcoholic, rarely, even in the most unhygienic surroundings, become phthisical." Raw[2] regards the gouty diathesis as antagonistic to phthisis, and he found that blood of gouty persons is not a suitable medium in which tubercle bacilli will flourish.

Cholelithiasis and Nephrolithiasis.—Persons with stone in the kidneys or gall-bladder only rarely develop active pulmonary tuberculosis. An attack of nephritic or biliary colic is only rarely observed in sanatoriums and hospitals for tuberculous patients. Among 128 subjects who came to autopsy from my service at the Montefiore Hospital, not one was found with stone in the kidneys, and only 1 with stone in the gall-bladder. We are at a loss to account for this mutual exclusion. The fact that stone is one of the manifestations of the arthritic and uric acid diatheses shows that it is in line with the rarity of phthisis in rheumatic and gouty subjects.

Atherosclerosis.—Patients with atherosclerosis, including aged persons, as well as those whose arteries calcify prematurely, only rarely suffer from progressive pulmonary tuberculosis. In some we find that the symptoms of tuberculosis are quite marked, especially the cough, expectoration and hemoptysis, which is probably more common than in tuberculous patients with elastic arteries, but the symptoms of toxemia are usually lacking; there is no fever, nightsweats, emaciation, etc. The lesion in the lung tends to fibrosis, and is often circumscribed and limited to one lobe.

When occurring in young persons, the cause of atherosclerosis is frequently syphilis, or interstitial nephritis; and both, as was already stated, are antagonistic to tuberculosis. Moreover, atherosclerosis is a manifestation of age, or even of senility, and tuberculosis in the aged usually pursues an intensely chronic course. It is the tendency to fibrosis and calcification, characteristic of atherosclerosis, syphilis, and senility, combined with a tendency to the deposit of calcium in the tissues, that probably retard the progress of the tuberculous lesion. Similar conditions are observed in interstitial nephritis and alcoholism.

Diabetes.—The traditional belief that diabetics are more liable to develop pulmonary tuberculosis than those with normal sugar metabolism has been somewhat shattered of late. S. Solis-Cohen even maintains that the number of cases in which tuberculosis supervenes upon diabetes is far less proportionately than the number of cases of tuberculosis found among persons whose carbohydrate metabolism is

[1] Lancet, 1904, **1**, 924.
[2] Tuberkulosis, 1911, **10**, 169.

not obviously disturbed, who, at all events, do not manifest glycosuria of glycouresis either persistent or permanent. In fact, the large percentage of diabetics presenting tuberculous lesions when examined at autopsy is not at all formidable when we recall that similar, or even higher, percentages are found when careful autopsies are made of persons who died from any cause. Thus, Montgomery[1] found that out of 355 autopsies collected from the literature since 1882, including his own 25 cases, 138, or 38.9 per cent revealed tuberculous lesions in the lungs, mostly in the acute form. This cannot be considered very excessive when it is borne in mind that tuberculous lesions are found in similar, or even higher, proportions among persons between twenty-five and fifty years of age. Joslin found among 1146 cases observed from 1894 till 1916 only 3.1 per cent with tuberculous lung lesions, which is lower than might be expected had their sugar metabolism remained normal.

Symptoms.—From the large number of diabetics with tuberculous lung lesions that have come under the writer's observation, it appears that the symptoms of phthisis do not differ materially from those observed in others. Some writers, notably Williamson, Magnus-Levy, and others, state that hemoptysis and also nightsweats are rare in diabetic consumptives. Most writers emphasize that in many cases the lesions are localized in the lower lobes of the lungs, and that tubercle bacilli may not be demonstrable in the sputum, all of which leads one to suspect that, in diabetics, the diagnosis of tuberculosis is made at times without a solid foundation. Personal experience in hospital and private practice has shown that there is no basis for the supposition that the symptoms and signs of phthisis differ materially in diabetics from those with normal sugar metabolism. In some, the course is progressive, in others, sluggish, while now and then we meet with fibroid phthisis which lasts for years without causing great inconvenience to the patient. I have met with cases in which both diseases lasted for over fifteen years. On the whole, it appears that tuberculosis is diagnosed in diabetics more often than facts would warrant.

The effects of tuberculosis on the metabolic disease is also of clinical interest. Magnus-Levy,[2] and other writers, have observed that with the appearance of symptoms of phthisis there occurs a decrease in the amount of sugar eliminated through the urine—in some patients it even disappears. Magnus-Levy says that this is due to the anorexia and cachexia characteristic of phthisis. But Naunyn observed that patients without these symptoms may show increased sugar tolerance with the onset of the tuberculous complication. It appears that only in mild cases is the increased sugar tolerance to be observed; in those with progressive tendencies, especially with acidosis, the elimination of sugar continues, as a rule, unabated. This point is to be borne in

[1] Am. Jour. Med. Sci., 1914, **147**, 866.
[2] In Kraus and Brugsch's Spez. Pathol. u. Therapie, Berlin, 1914, **1**, 35.

mind when judging the effects of any method of treatment of diabetes in tuberculous patients.

Rarity of Glycosuria in the Tuberculosis Patients.—It is noteworthy in this connection that while tuberculosis often complicates diabetes, the contrary is not true. Whenever the two diseases are associated, diabetes almost invariably preceded the tuberculosis. West, Raw, Montgomery, and many others, have reported this as their experience. In the writer's practice, dealing with thousands of tuberculous patients derived from a class peculiarly liable to diabetes (Jews), only 2 tuberculous patients were observed to develop glycosuria while suffering from active tuberculosis. Among 31,834 cases of tuberculosis, collected from 25 sanatoriums and hospitals in various parts of this country, there were 101 (about one-third of 1 per cent) cases of glycosuria and 51 (about one-sixth of 1 per cent) of diabetes, according to Landis, Funk and Montgomery.[1] Even conceding that some of the cases were undoubted diabetics, the percentage is exceedingly low. More than 1 per cent of the general population in this country is diabetic. Among 1047 autopsies made on tuberculous subjects only 6 were diabetics, and some of these may have been sick with diabetes before the development of the tuberculous lung lesion. In fact, among 373 tuberculous cases autopsied in the United States Army, not a single case of diabetes was discovered.

It is not clear why tuberculous patients never become diabetic. The fact that diabetes is a disease of excess in food and drink, while tuberculosis is one of undernutrition, may be suggestive, but it does not explain.

Prognosis.—In a large proportion of cases the tuberculous disease in diabetics runs a progressive course, leading to a fatal termination, which could be expected *a priori* considering that both are wasting diseases. But I have seen many in whom the tuberculous process was quiescent, or sluggish in its course, lasting for many years. Improvement may be seen in either, or both, diseases. It also seems that when the diabetes is of a severe character the tuberculous process is likely to pursue a progressive course, and *vice versa*. Next to acidosis, tuberculosis appears to be the commonest cause of death in diabetes. Social conditions have a great influence; among the well-to-do the association of the two diseases is not always rapidly fatal. Having seen healed tuberculous lesions in the lungs of individuals who succumbed to diabetes, the writer is inclined to the opinion that, while serious, the complication is not invariably fatal.

Obesity.—Pulmonary tuberculosis occurs very frequently in well-nourished individuals, but only rarely in the obese in the full sense of the word. The reasons are obvious: Tuberculosis, consumption, is the direct antithesis of obesity. But now and then we meet with a case of undoubted active phthisis in an obese person. As a rule, the

[1] Am. Rev. of Tuberc., 1919, **2**, 690.

tuberculous process tends to cicatrization in these fat patients. In some instances the pathological process in the lung proceeds from infiltration to softening and the formation of large cavities; but the excavations are, as a rule, circumscribed and surrounded by dense fibrous capsules. The patient keeps on coughing for many years, and expectorates sputum containing tubercle bacilli. But hardly any symptoms of tuberculous toxemia are observed, excepting perhaps dyspnea and cyanosis.

The prognosis in these cases of "fat consumption" is good, so long as there is no fever. But finally, if not carried off by some intercurrent disease, fever develops, emaciation becomes pronounced, and the patient succumbs with the usual terminal symptoms of phthisis. While this occurs in many cases, still it is a rare phenomenon. Fat consumptives live very long; some as long as might have been expected had they not developed tuberculous lesions in the lungs.

At times we observe patients who had always been of average, or below the average weight, develop tuberculosis; the lesion in the lungs heals by cicatrization and cavity formation, and then they become more or less obese. This is commonly seen in women over forty years of age, though rarely it occurs in younger persons, of either sex. The obesity in these cases is of the plethoric type, and they are liable to more or less copious pulmonary hemorrhages which usually give them relief for some time. Most of them have mild fever, as many obese persons do; they cough and expectorate, etc. Especially annoying is the dyspnea, due to both the obesity and the limitation of the breathing area in the lungs.

It appears that the obesity in consumptives is, in many instances, due to some dysfunction of the internal secretory glands, as is attested by the fact that it occurs frequently in women during the menopause, and in men with gonadal tuberculosis (see p. 610). But we also see it in patients who have been thoroughly hospitalized; who have spent years in various sanatoriums and acquired a knowledge of the great benefits to be derived from perfect rest, nourishing food, etc.

Dysfunctions of the Endocrine Glands.—The association of certain endocrine disturbances with pulmonary tuberculosis is quite frequently observed. Considering the great prevalence of tuberculosis, it may be assumed that in many cases the association is altogether coincidental. But in many instances it appears that one disease is either stimulated, or arrested in its progress, by the other. This is especially the case with dysfunctions of the thyroid, adrenals and the gonads.

Thyroid Dysfunction.—It is well known that quite a large proportion of tuberculous patients present symptoms of hyperthyroidism such as more or less bulging eyes, tachycardia, a slight tremor, hot flushes, sweating, dermographism, and even enlargement of the thyroid gland (see p. 290). This cannot be attributed to tuberculosis of the thyroid because it has been shown that this gland is relatively immune to the tubercle bacillus. Rokitansky never observed a case, and Virchow

said that no organ is so little disposed to tuberculosis as is the thyroid. To be sure, cases have been reported in which the thyroid showed tuberculous changes at the autopsy, or after surgical removal; in this country Mosiman[1] reported such cases. But, at all events, they are rare. Albert Kocher,[2] whose experience has been extensive, says that the thyroid displays a strong resistance against the virus of tuberculosis, and that tuberculous changes in the gland are observed only in extremely rare instances. When the thyroid, derived from tuberculous cases, is examined histologically the changes are those of sclerosis, as a rule, the direct antithesis of those found in Graves's disease. Kehl says that he could not find any tuberculous changes in the thyroid of 50 tuberculous patients.

It is not generally appreciated, but it is a fact, that in patients with symptoms of hyperthyroidism, tuberculosis, when it does occur, runs a very mild course; in many the abortive type of the disease is to be seen. In cases in which the differential diagnosis between hyperthyroidism and pulmonary tuberculosis has to be made, we are often actually dealing with the coexistence of the two diseases, but the lung lesion is so mild, the patient recovering, and remaining with the thyroid dysfunction, that we are apt to conclude that the suspicion of tuberculosis was not justified. In progressive cases of phthisis we may note symptoms of hyperthyroidism in the incipient stage, but with the advance of the tuberculous disease, they disappear. They may also disappear when the lung lesion improves, or when the patient is cured of his tuberculosis.

The transitory character of the hyperthyroid symptoms, disappearing with aggravation, as well as with improvement, in the tuberculous process, is rather suggestive. It appears that the tuberculous toxin stimulates the thyroid at first, but when the stimulation keeps on for a considerable time, it is effective in producing sclerosis of the gland, and for this reason we find sclerosis of the thyroid in many fatal cases of tuberculosis. Moreover, hyperthyroidism is often found in youthful tuberculous patients, in adolescents, in young girls in whom menstrual disturbances, dysmenorrhea, amenorrhea, etc., are clinical features of the case.

The mildness of tuberculosis in hyperthyroid individuals was observed fifty years ago by Hamburger,[3] and Morin[4] noted long ago that in tuberculous families the members who have large thyroids escape, and when infected, recover. S. Solis-Cohen stated as far back as 1887 that a large thyroid is characteristic of immune members of tuberculous families. Greenfield[5] could not find tuberculous lesions in any of the fatal cases of Graves's disease that came under his observa-

[1] Surg., Gynec. and Obst., 1917, **24**, 680.
[2] In Kraus and Brugsch's Spez. Pathol. u. Therapie, Berlin, 1919, **1**, 819, 989.
[3] Quoted from Muralt, Med. Klinik, 1913, **9**, 1814.
[4] Rev. Méd. de la Suisse Romande, 1895, **15**, 241.
[5] Lancet, 1893, **2**, 1554.

tion. Similar experiences have been reported by Bialokour,[1] Brandenstein,[2] and others. Among 45 cases of hyperthyroidism observed by Saathoff[3] all had signs of tuberculosis, but of a mild type, only 2 were of the "open" type. Turban,[4] Gerald Webb,[5] and many others have had the same experience. In Webb's[6] opinion "the increase in the size and function of the thyroid is a phase in the marshalling of the body's defensive forces against the invading disease." Webb quotes Plummer, of the Mayo Clinic, where large numbers of cases of exophthalmic goiter are treated, to the effect that tuberculosis is much more rare in persons with this disease than in others.

This is in agreement with our observations presented while discussing the status lymphaticus (see p. 588). It is well known that during adolescence some youths with symptoms and signs of the lymphatic diathesis grow rather fast in height, their hands and feet enlarge, thus presenting the stigmata of either dyspituitarism, or dysthyroidism (see p. 575). It is just at this period of life that pulmonary tuberculosis develops in many persons. It is noteworthy that "doubtful" cases are most frequent among adolescents, and that many of these "suspects" recover, to tell in later life the usual story that during their early life some doctor diagnosed tuberculosis in them. Fr. Kraus[7] and Julius Bauer[8] have pointed out that these individuals are liable to develop extra-pulmonary tuberculous lesions, but I cannot say from personal experience that such is the case.

Hypothyroid individuals, on the other hand, very frequently suffer from active and progressive tuberculosis of the lungs. In fact, in thyroid families some children are myxedematous, or cretins, while others are tuberculous. W. S. Greenfield speaks of "the great tendency in myxedema to tuberculosis. Of course, it may occur in Graves's disease, but as far as I can judge there is no special tendency to it. In myxedema it is especially frequent." In 5 out of 7 fatal cases of myxedema, tuberculosis of the lungs was found at autopsy; while in none of the cases of Graves's disease was tuberculosis found at autopsy. Greenfield suggests that perhaps thyroid medication may be helpful in these cases. Symptoms and signs of hypothyroidism are very often noted in far-advanced cases of tuberculosis.

Dysfunction of the Adrenals.—In a very large proportion of cases of pulmonary tuberculosis there are to be seen symptoms pointing to dysfunction of the adrenals. The low blood-pressure, as well as the weakness and lack of enduring powers, pigmentation of the skin, etc., have been attributed to hypofunction of the adrenals. The great frequency with which these glands are found affected by tuberculosis

[1] Ztschr. f. Tuberkulose, 1910, **16**, 230.
[2] Berl. klin. Wchnschr., 1912, **49**, 1840.
[3] München. med. Wchnschr., 1913, **60**, 230.
[4] Beitr. z. Kentniss der Lungentuberkulose, 1899.
[5] Jour. Labor. and Clin. Med., 1916, **1**, 414. [6] Am. Rev. Tuberc., 1921, **5**, 266.
[7] Ztschr. f. Tuberkulose, 1913, **19**, 417.
[8] Konstitutionelle Disposition zu inneren Krankheiten, Berlin, 1917, p. 361.

in cases of phthisis, favors the view that they are due to insufficiency of the adrenals. Many writers have seen a correlation between the excessive function of the thyroid in incipient tuberculosis and the insufficiency of the adrenals. On the other hand, Gerald Webb and his co-workers found hypertrophy and hyperfunction of the adrenals in experimental tuberculosis.

It is noteworthy that while in nearly 90 per cent of cases of Addison's disease tuberculous changes are found in the adrenals, active tuberculosis of the lungs is very rare in this disease. Most of the lung lesions found at the autopsy are of the sclerotic type. According to some authors, notably Kraus, Bauer, Bartel, and others, Addison's disease occurs mainly in individuals of the lymphatic constitution, and the lungs are thus shielded against tuberculous changes by the extrathoracic lesions, as we have already shown (see p. 584). In fact, tuberculous lesions in the lungs found in many cases of Addison's disease are almost invariably of slight extent and healed.

The Gonads.—We have already spoken of the rarity of active and progressive tuberculosis of the lungs in cases of tuberculosis of the male generative organs (see p. 587). In women also it appears that when tuberculosis occurs during the menopause, it is apt to run a sluggish course, frequently of the fibroid type. In several cases of tuberculosis of the female generative organs, verified by examination of the organs removed surgically, the lungs have been found free from active disease. I have also observed that in such cases, after successful operative removal of the tuberculous adnexa, active and progressive tuberculous disease of the lungs is likely to be of its appearance. Here again we find evidence that extrathoracic tuberculosis is to a certain degree antagonistic to pulmonary tuberculosis.

Many cases of tuberculosis in women over forty years of age show the effects of hypofunction of the gonadal organs more than those of the tuberculous toxemia. The lung lesion, as has just been stated, is likely to be sclerotic and give very little trouble. But symptoms due to hypofunction of the ovaries are pronounced. It is difficult to say whether the hyperfunction of the thyroid, characteristic of the menopause, is responsible, but it is a fact that these patients get along for many years though showing signs of active tuberculosis. The constitutional symptoms are very frequently relieved by the administration of organotherapeutic products, as will be shown later on.

The fact that fibroid phthisis is most common in persons over forty years of age, and that tuberculosis in the aged pursues, as a rule, a very sluggish course, are suggestive in this connection.

In contrast with the chronicity of phthisis in individuals in whom the function of the sex glands is below par, we observe that when the disease occurs in youthful individuals, especially at the age of puberty, when the function of the gonads is at its highest, the process is very often acute and progressive. In boys between sixteen and twenty-five years of age, acute pneumonic phthisis is very frequent, and when the disease

is of the chronic type, it very often shows tendencies to progression; soon after the onset we often find that an entire pulmonary lobe is involved. Remissions are short-lived. The general appearance of these patients is striking to the observant clinician because of the pathognomonic "facies" (see p. 299), the ghastly pallor which is quite common, and by the high fever and prostration. Arrest of the disease may occur, but not as often as in persons over twenty-five years of age. Such acute cases are rare among persons over thirty, and exceptional over forty-five, excepting, of course, miliary tuberculosis, which is the last straw in many cases of chronic pulmonary phthisis. One of the characteristics of phthisis at the age of puberty is the uncommonly high stature (see p. 576), which again points to hyperfunction of the gonads.

The relationship of phthisis to puberty is, however, best seen in girls. When fully developed, phthisis often results in amenorrhea. But it has been observed, first by H. Hanford,[1] that early or excessive menstruation in young girls is often associated with tuberculosis. He stated also that the female children of phthisical parents tend to menstruate unduly early, and excessive. More recently Scherer,[2] in a study of 10,216 cases of tuberculosis in women arrived at somewhat similar conclusions. He found that in girls who menstruate early, or excessively, the prognosis of tuberculosis is more grave than in those who begin to menstruate later and have either a normal or scanty flow. This may perhaps be correlated with the observation of botanists that sickly and weakly plants blossom earlier and bear more fruit, numerically, than healthy and vigorous ones. We have already referred to the fertility of tuberculous individuals (see pp. 298 and 579).

More recently this has been confirmed experimentally by Hans Mautner.[3] He found that when castrated guinea-pigs are infected with tubercle bacilli they live longer than normal controls, while thyroidectomized animals die sooner. He also reports that castrated animals, when infected, display strong tendencies to recovery, the lesions are localized; while the control animals become emaciated, the castrated ones showed strong tendencies to the deposition of fat on the body.

It is well known that the establishment of puberty has a more profound effect on girls than on boys, and that it occurs earlier in the former. And at this period of life, the mortality from tuberculosis is higher among females than among males. As can be seen from the table on page 85, the rates per 100,000 were among girls ten to fourteen years of age, 29.4, and among boys, only 12.2; between fifteen and nineteen years of age, girls, 110.9, and boys, 79.6. After twenty-five years of age, the mortality is higher in men than in women. Vital statistics in other countries are to the same effect (see Figs. 10 and 11).

[1] British Med. Journal, 1887, **1**, 153.
[2] Beitr. z. Klinik. d. Tuberkul., 1921, **40**, 7.
[3] Monatsschrift f. Kinderheilk., 1921, **21**, 38.

Gastro-intestinal Diseases.—The reciprocal relations between diseases of the gastro-intestinal tract and pulmonary tuberculosis have been discussed in Chapter XI. While ancient authors spoke of "gastric phthisis," it may be stated that we do not know of any disease of the stomach which predisposes to pulmonary tuberculosis, or is found in phthisical patients with sufficient constancy to make it pathognomonic of the disease. The anorexia of phthisis is easily explained by the fever, cough, expectoration, etc.; while the emetic cough, so often troubling phthisical patients, is not due to any structural or functional changes in that viscus. Gastric ulcer is at times found in patients suffering from tuberculosis, but not more frequently than in non-tuberculous. In 128 autopsies from my service at the Montefiore Hospital only 1 was found with an ulcer, and it was not of a tuberculous nature.

Intestinal ulcerations occur very frequently in tuberculous patients (see p. 554) but they are due to the same cause as the lung disease. There are, however, two intestinal diseases in which there have been observed certain relations to pulmonary tuberculosis; they are appendicitis and fistula-in-ano.

Appendicitis.—In many tuberculous patients with acute or subacute, or adhesive pleurisy of the membrane covering the lower lobe of the right lung, symptoms and signs not unlike those of appendicitis are often found. This is also true of ulcerations in and near the cecum, or the peritoneum covering that part of the bowel. It is for this reason that many authors have reported that a large proportion of tuberculous patients suffer from appendicitis requiring surgical intervention, while others have maintained that the contrary is true.

The number of patients with long-standing pulmonary tuberculosis who have abdominal scars indicating operations for appendicitis is high; for this reason also some authors have stated that appendicitis is an etiological factor in tuberculosis. Faisans, Walther, Siredey, Claisse, Thiroloix, and especially Sergent,[1] are of this view. Sergent believes that excessive meat eating, in which tuberculous patients often indulge is responsible. He suspects every patient with chronic appendicitis of tuberculosis. Letulle found that 30 per cent of consumptives present inflammatory lesions of the appendix, and Bialokur[2] maintains that from 25 to 30 per cent of all phthisical patients suffer from chronic appendicitis, and 50 per cent show tuberculous lesions of this rudimentary structure.

The clinician will become suspicious when he finds that most patients with symptoms of appendicitis show signs of pleurisy in the right side of the chest, especially the pleura covering the lower lobe is found inflamed or adherent. The referred pain in such cases have already been described in detail (see p. 483). It appears that those who carefully examine the chest for signs of pleurisy have very few tuberculous

[1] Études cliniques sur la tuberculose, Paris, 1919, p. 368.
[2] Internat. Zentralblatt. f. ges. Tuberkuloseforschung, 1915, **9**, 137.

patients who need operations for appendicitis. Gerald Webb[1] is of this view. At the Montefiore Hospital we only rarely see a case of appendicitis requiring surgical intervention among the tuberculous patients. The pain in the right iliac fossa often seen in tuberculous patients may also be due to tuberculous ulcerations of the cecum, or localized peritonitis in that region, and more commonly to diaphragmatic pleurisy.

Fistula-in-Ano.—The association of fistula-in-ano with tuberculosis has been noted for generations, and some have maintained that a fistula is a sure indication of an active lung lesion. Hartmann stated that 5 per cent of tuberculous patients have fistulæ, a proportion which is rather excessive; less than 2 per cent of patients coming under the writer's care have been found with anal fistulæ.

Many surgeons state that the vast majority of fistulæ are in themselves tuberculous. Among 197 cases reported by Melchior[2] 61 per cent were tuberculous; in Goetz's[3] series the proportion was 43 per cent, while other authors found it much less. Harvey B. Stone,[4] in a recent study of the question, hazards the estimate that probably in 15 to 30 per cent of all fistulæ cases a fundamental relationship with tuberculosis may be established. The difficulties in establishing the tuberculous nature of fistulæ lies in the fact that nearly all, in addition to tubercle, have also been infected with pyogenic microörganisms, and the result is that the characteristic microscopic structures are not easily discerned; obviously, because of mixed infection, inoculation tests are likely to prove misleading.

It has been the experience of the writer that most tuberculous patients with fistula-in-ano do well; the process in the lung is usually quiescent or latent, so as to render the diagnosis doubtful. For this reason, probably, the diagnosis is not made as frequently as facts would warrant. Moreover, it appears that after surgical intervention these fistulæ recur in a large proportion of cases. As long as the fistula keeps on discharging, the pulmonary lesion is likely to remain latent or quiescent. But in the comparatively few cases in which operation is successful in closing up the fistula, the process in the lung is likely to become active and progressive. I have seen numerous cases of this sort. Melchior's statistics bear out this point. For this reason it has been my practice recently to discourage operations for fistula-in-ano, so long as it is not unbearably troublesome. In most cases operated on, or when the fistula closes up, the pulmonary lesion flared up within a few months or years. Similar effects of operations on other extrathoracic tuberculous lesions have already been mentioned.

Cirrhosis of the Liver.—Many writers have spoken of a frequent association of hepatic cirrhosis with pulmonary tuberculosis. It

[1] Tr. Nat. Tuberc. Assn., 1916, **13**, 202.
[2] Beitr. z. Klinik d. Chirurgie, 1910, **70**, 475.
[3] Inaug. Diss., Tübingen, 1916.
[4] Am. Rev. Tuberculosis, 1917, **1**, 548.

appears that many patients suffering from cirrhosis succumb finally with symptoms of pulmonary, and at times, peritoneal tuberculosis. According to Rolleston,[1] the cause of death in hepatic cirrhosis is pulmonary tuberculosis in 12 to 14 per cent of cases. He compiled statistics of 584 cases of cirrhosis obtained by adding together the figures in the statistics of Lancereaux, St. George, Kelynack, and Yeld, and showed that tuberculous lesions were found in the lungs in 132, or 22.6 per cent.

In pulmonary tuberculosis the liver is affected very frequently; in fact, in a very large proportion of fatal cases miliary tubercles are found in the liver; in others, tuberculous abscesses, due to focal necrosis; fatty degeneration is seen in the vast majority of fatal cases, and amyloid changes in those who, for a long time, suffered from suppurating and cavitary lesions in the lungs or other organs. Several French authors, notably Hanot and Gilbert, have maintained that cirrhosis is often found in patients who died from pulmonary tuberculosis, and they also brought some experimental evidence to the effect that tubercle bacilli may be instrumental in inducing hepatic fibrosis with resulting destruction of the parenchymatous cells. However it appears that this is exceptional, and even when occurring in far-advanced stages of phthisis, it is of little clinical importance, for obvious reasons. That it is rare, is seen from Lorentz's statistics of 4337 autopsies, among which 111 were cases of cirrhosis. He found that of those who died from cirrhosis, 20 per cent had tuberculous lesions, while those who died from tuberculosis showed hepatic cirrhosis in only 2 per cent of cases.

Cancer.—Ever since Rokitansky, in 1841, observed what he considered a distinct antagonism between tubercle and cancer, many authors have brought forward evidence in substantiation of this contention, while others have denied it. Rokitansky found that organs which are liable to cancerous infiltration, as the ovary, the salivary glands, the stomach, esophagus, rectum, etc., are only rarely affected by tubercle; conversely, organs frequently showing tuberculous changes, as the lungs, the ileum, etc., are only rarely affected by malignant neoplasms.

Autopsies performed on large series of cases seem to substantiate Rokitansky's opinion. Thus, Otto Lubarsch[2] reported 6536 autopsies, among which 2668, or 40.8 per cent, were found tuberculous, and 569, or 8.7 per cent, carcinomatous. Of the tuberculous series 117, or 4.4 per cent, were carcinomatous; while of the 3868 non-tuberculous, 452, or 11.7 per cent, were cancerous; of the 569 cancerous, 117, or 20.6 per cent, were tuberculous, while of the 5967 non-cancerous, 2551, or 42.7 per cent, were tuberculous. Among 1445 necropsies of persons who died from cancer compiled from the literature by Broders,[3] only 236, or 16.3 per cent, showed associated tuberculous lesions, which,

[1] Diseases of the Liver, 1905, pp. 221, 281.
[2] Virchows Archiv, 1888, **111**, 280.
[3] Jour. Am. Med. Assn., 1919, **72**, 390.

compared with the high percentage found in the general run of autopsies, ranging usually upward of 50 per cent, shows that the two diseases are not commonly found in the same individual. Landis[1] reports 633 autopsies performed at the Phipps Institute, all tuberculous, and among which there was not a single case in which there was associated cancerous growth. Roger Williams[2] found tuberculous lesions only twice in 136 cancer autopsies, and he quotes Kelynack, of the Manchester Infirmary, who found it only twice in 145 similar autopsies. Likewise, McCaskey[3] making autopsies on 281 cancer patients found tuberculous lesions only in 1.25 per cent; in the non-cancerous its frequency was nearly twenty times as great. James Kingston Fowler[4] also states that it is very rare indeed to find tuberculosis and cancer in active progress at the same time.

We do find, however, in many cases of fatal cancer evidences of tuberculous lesions in the lungs at the autopsy. These are mainly of two varieties: In most cases the tuberculous lung changes are sclerotic, or calcified, practically healed; in others, especially in those who died from malignant neoplasms of the intrathoracic organs, pressure of the tumor on the contiguous parts of the lungs stimulates the reactivation of dormant tuberculous lesions. The writer has seen 2 cases of cancer of the lung with old tuberculous cavities, which, however, remained inactive while the neoplastic process progressed. In 2 cases of cancer of the lung, verified by autopsy, tubercle bacilli were found in the sputum.

The reason why there is a certain degree of antagonism between tuberculosis and cancer is not known at present with our meager knowledge of the etiology of malignant neoplasms. The suggestion has been made that the age incidence of the two diseases is responsible, tuberculosis occurring mainly before thirty-five, and cancer above that age. But this does not hold, because tuberculosis is not at all uncommon in persons above forty years of age, and it is in these persons, of the cancer age, that tuberculosis only rarely develops once they have malignant tumors.

There is hardly anything of interest about the clinical manifestations in the rare cases in which the coexistence of the two diseases is discovered. At times when a patient with carcinoma of the bronchi or lung develops tuberculosis, tubercle bacilli may be found in the sputum, and thus some doubt is cast as to the correctness of the diagnosis of pulmonary neoplasm, as had occurred in 2 cases under the writer's care. But it appears that the severity of the symptoms of cancer obscures the symptoms of tuberculosis, and in due time either metastasis, or some other feature of the case, clears up the diagnosis.

[1] Quoted from Hoffman, The Mortality from Cancer Throughout the World, Newark, 1915, p. 188.
[2] Twentieth Century Practice, 1908, **17**, 270.
[3] Am. Jour. Med. Sci., 1902, **124**, 97.
[4] Diseases of the Lungs, London, 1898, p. 316.

Insanity.—For generations it has been observed that the tuberculosis morbidity and mortality in insane asylums is exceedingly high; in some as much as three or four times that of the general population. This fact, in connection with the peculiar psychology of the consumptive, has led some authors to see a correlation between tuberculosis and mental alienation.

It has been an almost universal observation that mental defectives are more liable to develop tuberculosis, and to die as a result of this disease, than the average population. This is particularly true of inmates of institutions for feeble-minded, in which more than 50 per cent of deaths have been due to pulmonary tuberculosis. Among the insane about 30 per cent of deaths are due to this cause. Those suffering from the depressive psychoses, and dementia precox, are most likely to contract phthisis.

As far as we know it is not insanity *per se* that is the predisposing cause to tuberculosis. When we bear in mind the mental condition of the insane, the overcrowding in the institutions, the carelessness of the patients as regards food and personal hygiene, it is not surprising that many contract the disease in whose etiology these factors play such a great rôle. This is confirmed by the high tuberculosis mortality in asylums in backward countries, where the hygienic surroundings, and food given the patients, leave much to be desired. In England and Wales it was observed that during the World War, when the administrative personnel of the asylums was depleted, the mortality from tuberculosis showed an appalling increase; among the female patients the increase was not as large as among the males, for obvious reasons. On the other hand, in this country, while quite high, the tuberculosis morbidity and mortality are not as high as observed in European institutions. Thus among 962 cases of psychosis coming to autopsy in the Manhattan State Hospital for the Insane in New York, active pulmonary tuberculous lesions were found only in 131 cases, or 13.60 per cent (Irving J. Sands). This low incidence may be ascribed partly to greater diligence on the part of the pathologists in searching more for changes in the nervous system than in the lungs, yet, even so, it is much lower than has been observed in other institutions. Perhaps the better care and food are responsible.

The diagnosis of tuberculosis in the insane is rather difficult in many cases. A mental defective does not complain of the mild symptoms characteristic of incipient tuberculosis, and in institutions the disease is discovered mostly when it is far advanced. When diagnosed, it is difficult to institute proper treatment in mental defectives because of lack of coöperation on the part of the patients.

While tuberculosis is common in the insane, the reverse does not hold. Insanity is rare among tuberculous patients. The psychic manifestations of the tuberculous, which have already been referred to (see p. 294), are mainly of toxic origin, due to the constant absorption of toxins from the tuberculous lesions. But some authors have seen

in this a sufficient cause for limiting the legal responsibility of tuberculous patients (Baccelli, Saxe, Jessen, Microli).[1] They point to the fact that the psychic state of the average consumptive in the advanced stages of the disease shows marked divergences from what we are wont to consider normal. The oscillating emotional manifestations, the alternating moods, changing quickly from supreme exaltation to extreme depression, and the lack of appreciation of the true condition even during the most ominous periods of the disease, show that the mind of the tuberculous patient is not in equilibrium with his withering body. Some have drawn a striking analogy between the consumptive who is in a state of intoxication by the toxins of the tubercle bacilli and decayed pulmonary tissue, and the alcoholic. Indeed, the external appearance of the consumptive, walking around in a febrile state, is somewhat similar to that of one under the influence of alcoholic intoxication. The bright eyes, the dilated pupils, the flushed cheeks, remind one of alcoholic intoxication, as Microli has pointed out. Mentally, also, there are great similarities: The flickering intelligence, which brightens up suddenly for a few hours soon to be followed by mental depression; the impulsive actions, the perverse stubbornness and egotism, all of which, at times, lead to criminal, or antisocial acts, combined with the quick transitions from optimism to pessimism, are seen among both alcoholic and far-advanced tuberculous patients. That these are mainly due to the tuberculous toxemia is shown by the fact that injections of tuberculin have been observed to either engender, or to accentuate these psychic conditions.

For these reasons, alienists who are in favor of limiting the legal responsibilities of those who are so far deluded as to become incapable of estimating facts of their true relations, insist that antisocial acts of consumptives should be condoned. However, it is a fact that true insanity is extremely rare in tuberculous patients. Working in a hospital among patients derived from an ethnic stock peculiarly prone to mental alienation, the writer has only twice in twelve years sent patients to insane asylums. In private practice also very few of the tuberculous patients under the writer's care became insane.

[1] For literature see Jessen, Lungenschwindsucht und Nervensystem, Jena, 1905.

CHAPTER XXXI.

PROGNOSIS IN PULMONARY TUBERCULOSIS.

The Curability of Phthisis.—Laennec, the first physician to make a scientific study of the pathology of phthisis, and who really introduced physical diagnosis, pronounced it an incurable disease. It appears, however, that this keen clinician recognized that many cases do recover. He said: "The cure of phthisis is not beyond the powers of Nature, but it must be admitted, at the same time, that art possesses no certain means of attaining this end."

The observations of physicians all through the nineteenth century have clearly shown that phthisis is not invariably fatal, despite the fact that the treatment applied during the first half of the nineteenth century should have killed most of the curable cases, according to our understanding of the pathology and therapy of the disease. Still, Flint reported 670 cases observed during a period of thirty-four years, and the proportion of cases cured or arrested was not much below that which we attain at present. Thomas J. Mays[1] compiled statistics of Flint's 670 cases and Williams's 1000 cases observed for twenty-two years, and compared the results with Trudeau's 1060 cases under observation for seventeen years. The percentages of recoveries and survivals are about the same, or rather in favor of Flint's and Williams's cases.

At present *we have sufficient and incontrovertible proof that tuberculosis is curable in all its stages.* Experience while making autopsies shows, in fact, that *it is the most curable of chronic diseases,* considering the enormous number of persons who show healed, or quiescent, tuberculous lesions in the lungs when examined after death. And the lesions discovered are often such as to indicate that the process was quite extensive at the time of its activity.

Importance of Prognosis.—There is no need of elaborating on the importance of prognosis in the practice of medicine. It is always significant and, in the case of tuberculosis, it is, at times, even more important than diagnosis. Indeed, most patients come with ready-made diagnoses and all they want to know is the ultimate outlook. "Will he recover?" is one of the first questions after the patient and his friends are told that there is a tuberculous lesion. "If so, how long will it take till he recovers?" Moreover, it is important to be ready to answer whether the patient, after recovery, will be able to resume his occupation, and whether there is danger of relapse. In case of an

[1] New York Med. Jour., 1914, **100**, 70.

unfavorable prognosis it is often asked, "How long will the patient last?"

We cannot answer all or most of these questions in the average case with a high degree of certainty. As J. Mitchel Bruce[1] says: "Prognosis in tuberculosis is always a difficult and often a disappointing proceeding. With all the facts of a case in our possession the conclusion we reach proves too frequently to be false. Indeed, paradoxical as it may appear, we fail in prognosis most often because of the very number, variety, and different character of the facts that we discover. Each of our observations has its own prognostic value, and most of them have a different value in different instances and at different times. We meet with an extraordinary, variable, and therefore uncertain, course of the pathological process from month to month. No disease is so difficult to deal with in this connection, and we have to confess that we too often find ourselves changing our forecast in both directions from time to time." The extreme difficulty of prognosis in phthisis has been best expressed by the one who said that he who attempts to forecast the outlook may be sure of one thing only, and that is that he will be mistaken.

The difficulties are, however, not insurmountable in many cases, and we can estimate the prognosis of the average patient in any stage of the disease with a certain degree of exactitude. But in order to do this, we must take into consideration all available facts which may have any bearing on the course of the disease.

Elements of Prognosis in Phthisis.—The notion that this disease is curable only in its incipient stage is one of the half-truths which have gained universal credence because of tradition. There are so many exceptions as to almost nullify this ancient dictum. We have already shown that it is fallacious to classify phthisis into three or four stages, and to say, without reservation, that in the first stage it is curable; in the second stage the chances of recovery are considerably diminished, while in the third stage it is incurable. *There are "incipient" cases which have no chance, irrespective of the treatment applied; while there are many in the third stage whose chances of survival and even of efficiency are excellent.* For this reason we shall not discuss the prognosis of phthisis according to the stages of the disease.

The elements of prognosis in phthisis reside in the following factors: (1) The form of the disease; (2) in a given form of the disease, the activity of the process as revealed by the constitutional symptoms and physical signs; (3) the presence of complications; (4) the extent of the lesion in the lungs; and (5) the economic condition of the patient.

Prognosis in the Various Forms of Pulmonary Tuberculosis.—We have seen from our study of the symptomatology of phthisis that the form of the disease has a greater influence on the ultimate outlook than the extent of the lesion, or even the activity of the process. Thus,

[1] Lancet, 1913, 1, 591.

in the pulmonary form of miliary tuberculosis, the chances of recovery are *nil*. The patient will die irrespective of the treatment applied. In acute pneumonic phthisis the prognosis is very unfavorable, the only hope we may entertain is that the disease will take a turn to the better, and pursue the course of chronic phthisis. This happens in rare instances, but it should not be expected in the average case. In fact, we may say that the prognosis is decidedly bad in these cases. *Patients with acute phthisis usually last as many weeks or months as those with chronic phthisis last years.*

On the other hand, taking the other extreme, abortive tuberculosis, we find that the prognosis is favorable under all circumstances. Practically all patients recover; the vast majority without even knowing that they have been tuberculous; or when the disease has been diagnosticated there often remains a lurking suspicion that it was a false alarm, even if tubercle bacilli were discovered in the sputum.

In fibroid phthisis the prognosis is very good indeed, so long as there is no fever. The dyspnea and discomfort which this disease causes for years are bearable by the average patient. But as soon as fever makes its appearance and persists for some time, the prognosis is that of chronic phthisis, which will soon be discussed.

The most important form of phthisis, that of the most common chronic type, is the disease in which the prognosis is very difficult to formulate in the individual case. We may be able to prove statistically that a certain percentage of cases recover completely; another percentage will survive so many years; still another percentage will succumb within one or two years, etc. But in the practice of medicine we deal with individual cases and statistics often count for naught.

In the individual case the outcome of the disease depends on so many complex and variable factors that it is often very difficult to formulate a prognosis. Indeed, we see that the most desperate case, slowly or suddenly, with or without any discoverable reason, takes a turn to the better and recovers. We see others who drag along for years; living, but they do not recover. Still others, in whom the general condition has been quite or altogether favorable, suddenly take a turn to the worse and the patient is carried off within a few weeks or months.

For these reasons we must enter into the elements of prognosis of chronic phthisis in greater detail.

Age.—The prognosis in children and in the aged has already been discussed (see pp. 471 and 477). In adolescents active tuberculosis of the lungs is very serious in most cases. If the lesion is extensive, and it becomes so in many cases, the outlook for recovery is not bright, though not hopeless, in most cases. Especially is this true in tuberculous girls under twenty years of age in whom the disease was preceded, or is accompanied, by amenorrhea. On the other hand, in women during the menopause the prognosis of tuberculosis is favorable. They may remain sick for many years, but they survive for a long time,

and may even be fit for ordinary housework, despite the activity of the process in the lungs. The common forms of chronic phthisis occur mainly during the ages of twenty to forty-five, and the prognosis is to be formulated in them by the criteria given in this chapter.

Prognostic Significance of the Patient's History.—Many authors have stated that patients with a family history of tuberculosis are more likely to run an unfavorable course than those derived from non-phthisical stock. A consideration of the facts brought together in Chapter XXX will show that this is a fallacious view. The patient was undoubtedly infected during childhood. Had he suffered a massive infection during infancy he would have succumbed to some acute form of tuberculosis. The fact that he survived the primary infection proves that it was mild; this is also the reason why he now suffers from chronic phthisis, and not from an acute form of the disease. Indeed, patients showing signs of some local tuberculous lesion at an earlier age usually have a slow, sluggish form of phthisis, lasting for many years. (See p. 584.) Many authors have calculated that the average duration of a phthisical patient with a family history of tuberculosis is longer than of one derived from robust stock. Recent medical literature abounds in statistics to this effect. Thus, Noel D. Bardswell,[1] in his statistical study of the patients at the King Edward VII Sanatorium, arrives at the conclusion that "having contracted tuberculosis, patients with a consumptive family history enjoy the same chance of recovery as those who have no such history." E. Arnould[2] shows that "a bad prognosis is relatively infrequent in patients with tuberculous parents—that is those who have had opportunities in infancy of receiving repeated small doses of infection; it is still rarer in those who have themselves previously suffered from some clinical manifestations of tuberculosis." The same experience has been recorded by Hayek[3] from statistics gathered in the German army. This is also seen in the acuteness of phthisis in persons who have just emigrated from rural districts into large cities (see p. 74).

Experience teaches that the prognosis is not different in tuberculous adults who are derived from phthisical stock than in those who are not. The slight differences that have been discerned appear to be rather in favor of the former.

Sex.—It appears that *the prognosis is more favorable in women than in men.* A man acquiring tuberculosis is apt to continue working and thus aggravate the prognosis while a woman, who is usually not the bread-winner, is more likely to abstain from overexertion, which is such an important element in the treatment of this disease. On the other hand, pregnancies, labor, and lactations are apt to aggravate the prognosis in women. But this factor has been overestimated. It appears that the mortality from tuberculosis of women during the

[1] Med. Research Committee, Special Report Series, No. 33, London, 1919, p. 65.
[2] Presse Médicale, 1921, **29**, 52.
[3] München. med. Wchnschr., 1919, **56**, 1316.

childbearing period of life, fifteen to forty-five, is lower than that of men of the same age (see p. 86). Married women are better cared for; their husbands work and provide for them, while when the husband—the breadwinner—is tuberculous, poverty is inevitable in many cases. For these reasons sanatoriums fill more of the male than of the female beds. Women are less likely to succumb to some of the more serious complications of phthisis, such as hemorrhage, pneumothorax, etc. They also less often suffer from laryngeal tuberculosis.

The Onset of the Disease.—*In cases with a sudden onset the prognosis is worse than in those in whom the disease came on insidiously.* Even the fact that the former are more apt to take strong measures to prevent the activity of the process does not counterbalance the seriousness of an acute onset, excepting when the suddenness refers merely to an initial pulmonary hemorrhage. An acute onset means severe constitutional and toxic symptoms, low powers of resistance, and the process in the lungs extends very quickly, so that in a short time quite large portions of one or both lungs are affected.

Those beginning with hemoptysis have usually a better outlook than others. The reason is not clear. Perhaps the dramatic onset frightens the patient, and he is apt to institute proper treatment even if he feels well after the cessation of the bleeding, while patients with mild symptoms, but without hemoptysis, may continue at work till the disease is aggravated. But this does not explain all cases. It seems that hemoptysis has very often a good influence on the prognosis of phthisis at any stage of the disease and many patients feel much better after a brisk hemorrhage (see p. 254). The cases marked by an onset with pleurisy, dry or moist, have, as a rule, a better prognosis than others, as has already been stated (p. 515). It has been observed that patients who are only slowly regaining their health after an attack of pleurisy and are pale and emaciated, are more likely to develop active and progressive phthisis than those who recover quickly, and soon regain their former health.

Prognostic Significance of the Activity of the Disease.—We have seen throughout this book that *the activity of the process in the lung has a greater influence on the ultimate outcome than the stage of the disease.* The activity is best studied by a careful consideration of general, or constitutional, symptoms. Of these, fever is the most important. There is no active tuberculosis without pyrexia. The afebrile cases, discussed elsewhere, are rather uncommon and it is a fact that the prognosis is rather good, so long as fever is lacking. Each turn for the worse, each complication, is accompanied by a rise in the temperature.

In active disease *the prognosis is unfavorable in direct ratio to the height and duration of the fever.* Every extension of the lesion manifests itself by increased pyrexia; persistence of pyrexia, despite rigid rest in bed, is pathognomonic of low resistance; the reverse type of fever, in which the highest point is reached in the morning instead of in the

afternoon or evening, is of grave prognostic significance—it may be an indication of an invasion of both lungs by tubercles. On the other hand, moderate fever, less than 101° F. dropping down to normal or subnormal in the morning, is rather favorable. In other words: *The higher the morning temperature, the nearer it approaches the evening temperature, the worse the prognosis.* Hectic fever, with normal and subnormal temperature in the morning, but which rises high in the afternoon and evening, is of grave prognostic significance. If it lasts for more than a month, the patient will not survive. He may last, or even improve for a time, but he will not recover.

A normal temperature throughout the day and night is a good sign; when accompanied by a good appetite, gain in weight, diminution in the cough and expectoration, etc., it is an indication of healing of the lesion. If fever only ensues after exertion or excitement, the prognosis is very good indeed, provided proper treatment is instituted. It is for this reason that most who have new and "infallible" remedies for phthisis ask for just this sort of cases on which to try the treatment. The vast majority recover under any treatment, provided good nourishment and rest are part of the "cure."

Indeed we can, in most cases, formulate our prognosis by a careful study of the temperature curve for a few weeks. Of course, we may on rare occasions err by putting implicit faith in the temperature curve, but the proportion of errors will be less than when we attempt to formulate it on other data, especially on the stage of the disease, or the findings on physical examination.

For this reason, *a prognosis in phthisis should not be given after a single examination of the patient.* It is required that the temperature of the patient should be studied for at least two weeks before attempting to forecast the outlook.

The prognostic significance of the pulse should be considered. *Excepting in heart disease and hyperthyroidism, no disease can be evaluated prognostically with the same degree of accuracy by the pulse-rate as chronic phthisis.* Incipient cases with a pulse not above 80 per minute have an excellent outlook. Tachycardia is an indication of acuteness of the process, or low resistance, or both. Patients who have apparently recovered but remained with a rapid pulse have a very poor outlook. The outlook is good in chronic cases with slow pulse.

Of the other constitutional symptoms which give us prognostic hints, the state of the gastro-intestinal tract is of great importance. Patients with good appetite and who digest and assimilate their food well, recover, even when they have, for the time being, some fever every afternoon. Persistent anorexia and gastro-intestinal disturbances are of grave prognostic significance. Gain in weight in afebrile patients with good appetite is a good sign. But occasionally we meet a patient who holds his own, or even gains, despite the fever. In such cases the thermometer should be our guide, and not the scale.

It has been the experience of sanatoriums that patients with positive sputum are more likely to have relapses of the disease after their discharge than those whose sputum has been negative, or became so after treatment. Thus at the King Edward VII Sanatorium, Bardswell's statistical studies showed that "the mortality figures indicate that cases in which tubercle bacilli have not been demonstrated in the sputum enjoy a substantially better prospect than those in which diagnosis has been confirmed by this 'positive' bacteriological find." While this favorable showing may be attributed to the fact that among the "sputum negative" cases a considerable proportion have not been tuberculous at all, and a still larger proportion have had abortive lesions, still, there is no doubt that the chances of improvement and survival are larger in sputum negative cases than in those with demonstrable tubercle bacilli.

Hemoptysis has no influence on the course and prognosis of the disease in the vast majority of cases. The initial hemoptyses are rather salutary, as was stated above. In the writer's experience, no patient has succumbed to a really initial hemoptysis. Ninety-eight per cent of cases of advanced disease recover from hemorrhages. But in cavitary cases, which may or may not be doing well, a brisk hemoptysis may unexpectedly kill the patient. In the individual case, if the hemoptysis is not accompanied by fever, or the fever lasts only a few days after the cessation of active bleeding, the prognosis is good. But if pyrexia continues it may point to acute pneumonic phthisis, or to tuberculous bronchopneumonia, which is almost invariably fatal. In these cases the hemoptysis is indirectly responsible for the fatal issue.

On the whole, however, patients with hemoptysis have not as good an outlook for recovery as those who never bled. Thus, from the statistics of Elderton and Perry it appears that "as a whole, the mortality among incipient cases which had had hemoptysis was considerably heavier than among the cases who had not had it, but in the advanced cases the excess was less. Hemoptysis seems to increase the mortality in case of male lives, and decrease it in the case of females; the difference in the latter case is; however, slight." The statistics gathered at Midhurst by Bardswell and Thompson show the same results, but they also point to "an initial hemoptysis may be associated with a relatively less severe type of the disease."

The *blood-pressure* of the patient may give us some valuable prognostic hints. Those with hypertension have a better outlook for recovery than those showing hypotension. Low blood-pressure is characteristic of feeble heart action due to the tuberculous toxemia acting unfavorably on the cardiac muscle. So long as the blood-pressure remains low, the prognosis is serious. With the improvement in the general condition of the patient there is almost invariably noted an increase in the blood-pressure. When there appears during the course of phthisis an abnormally high blood-pressure, an examination of the urine may disclose the presence of albumin and casts. In

fibroid phthisis, and in some cases of phthisis in gouty and rheumatic individuals, the blood-pressure is normal or above normal and the prognosis is good.

Prognostic Significance of Complications.—The presence of complications, tuberculous and others, modifies the prognosis perceptibly. Thus, laryngeal and intestinal tuberculosis aggravate the prognosis. Though many recoveries are seen in patients with these affections, yet in the individual case we must not give a favorable prognosis in those who show positive proof of laryngeal or intestinal complication. With advanced laryngeal disease, manifesting itself in aphonia, dysphagia, etc., a fatal issue is to be expected. The same is true of diarrhea, which lasts more than a month. We occasionally, however, see patients with profuse diarrhea lasting for several months. But they never recover. Blood in the stools is another unfavorable sign. Ischiorectal abscess is itself an indication of intestinal tuberculous ulceration and is of unfavorable prognostic significance.

Pleurisy is not invariably an unfavorable complication. The dry form occurs in nearly all chronic cases and has a rather salutary influence on the pulmonary lesion; it is also a good preventive of spontaneous pneumothorax. Pleural effusions are serious, though in many cases they have a good influence on the basic disease. We have already shown that they occasionally promote the healing of the lesion in the lung by compression. But in bilateral lesions the side with a free pleura is likely to suffer from an extension of the tuberculous process and the outlook is gloomy.

Empyema is a very bad complication. No recovery is to be expected. The patient may last for months, but he will not recover. In exceedingly rare instances the pus breaks through a bronchus and is expectorated. But even here the ultimate outlook is bad, because of the amyloid degeneration of the viscera, and the general malnutrition caused by the prolonged suppuration.

Spontaneous pneumothorax is fatal in 95 per cent of cases within one month of its occurrence. The exceptions have already been mentioned.

Tuberculosis of the kidney is of unfavorable import.

Of non-tuberculous complications we may mention influenza. This disease is more often diagnosed in tuberculous patients than facts would warrant. An increase in the cough, pyrexia, etc., due to an exacerbation of the tuberculous process, is apt to be attributed to influenza by patients and physicians. We have already discussed the influence of pneumonia on the prognosis of phthisis (see p. 590).

We often meet other non-tuberculous diseases in patients suffering from phthisis. Such as necessitate an operation with the administration of a general anesthetic are dangerous, and it has been my rule to urge local anesthesia, whenever feasible, in operations on tuberculous subjects. But when a general anesthetic is imperative, the outlook is not so grave as popularly supposed. Many tuberculous patients

under my care have been operated upon and held under the influence of ether or chloroform for more than an hour, yet they did well after recovering from the operation. In most cases the lesion in the lung keeps on pursuing its course as if no surgical interference had been instituted. C. D. Parfitt reports that 5 per cent of his sanatorium patients during seven years had to undergo major surgical operations with general anesthesia. Despite the surgical shock and anesthesia, the pulmonary condition was not aggravated in any case. Similar experiences are reported by H. G. Wetherill, of Denver, and H. M. Kinghorn. It seems that the entire problem rotates around the activity of the pulmonary lesion. An anesthetic administered to a patient with extensive lesions in the lungs, running high fever, having a rapid pulse, and other symptoms of tuberculous toxemia, will but accelerate the inevitable, or aggravate the slight chances of improvement. But when the lesion is quiescent, the temperature and pulse around normal, and the general condition fair or good, the patient will stand the shock of a major operation with general anesthesia.

Prognostic Significance of Signs Found on Physical Examination.—We have already mentioned the fallacy of formulating the prognosis of phthisis solely on the findings by physical examination. There are cases showing physical signs indicating that we are dealing with incipient, or first-stage, cases of the American or Turban classification, yet the prognosis is very unfavorable. Indeed, *the most unfavorable prognosis should be given in cases showing marked constitutional symptoms which are out of proportion to the findings on physical examination.*

It may be stated that generally the extent of pulmonary involvement is of more importance than the stage to which the lesion has advanced. Cavitation in one lobe is of less danger than infiltration of two or three lobes. J. Edward Squire gives the following table embracing 2720 cases of phthisis showing the relation of improvement to the number of lobes involved:

Lobes affected.	Cases.	Much improved. Per cent.	Improved. Per cent.	Total improved. Per cent.
1	877	58.38	28.62	87.00
2	1015	37.83	34.67	72.50
3	515	22.52	35.53	58.03
4	277	15.16	29.24	44.40

The fear and apprehension entertained by both the profession and the patient for "holes in the lung" are based on misconceptions of the pathology of phthisis. The fact is that the most dangerous cases of progressive phthisis are fatal before cavities are formed. This is the case with miliary tuberculosis and, to a certain extent, with acute pneumonic phthisis. If a tuberculous lesion in the lung does not cicatrize quickly, the best that can happen to the patient is that a cavity should form. *A pulmonary cavity is proof that the organism is in possession of strong powers of resistance, in fact, of immunity;* otherwise the lesion would spread. The difference between active phthisis with

cavity formation and without such occurrence is analogous to that between general septicemia and abscess. In the latter case the disease is localized and circumscribed and, when drained, the danger is not very great. A cavity has in fact, been defined as a tuberculous abscess which is drained through a fistulous opening into a bronchus.

This is a fact which is not appreciated at present to the extent it deserves, though over one hundred years ago that keen clinical observer Laennec already spoke of it. He said: "Pretty often, at the period when the complete evacuation of a tuberculous cavity is indicated by the stethoscopic signs, the patient experiences a marked improvement in his symptoms: the expectoration and fever decrease, and, if the improvement only last a little while, even the wasting of the body is sometimes diminished. This false convalescence is usually only of a few days' or weeks' duration; but it may extend to some months, and may even seem to be complete. . . . It may even, in some rare instances, terminate in a perfect and permanent restoration of health."

It may be stated that *the dangers of tuberculous cavities vary inversely with the time it takes for their formation.* The sooner they are produced, the worse the prognosis; the slower they develop, the better the ultimate outlook. In very acute forms of phthisis cavitation is very rare. The prognosis is gloomy with or without localized destruction of pulmonary tissue. In adults such cases are comparatively rare, but in infants rapid cavity formation is seen at times, and the termination is almost invariably fatal. In subacute forms of phthisis, in which excavations are apt to form very rapidly, the prognosis is unfavorable, unless the cavity is rather small. In the latter case the disease may be attenuated, and subsequently pursue a chronic course with the sequestration and expulsion of the affected area. Excavation is then the first step toward the diminution of the acuteness of the process in the lung. The general symptoms may be ameliorated, as after the evacuation of an abscess.

In chronic phthisis excavations, even when extensive, are compatible with a long and efficient life. These cavities are surrounded by more or less dense fibrous capsules which limit their extension, and are drained through fistulous tracts communicating with bronchi. So long as the secretions are eliminated by expectoration, the patient may feel quite comfortable for years. The cavities may even heal, as was already shown (see p. 178). When small, they may be obliterated by granulations or by calcification of their contents. Larger excavations may shrink, or even when remaining of large dimensions, they may become altogether benign after the necrotic tissue has been expelled. They are, however, a constant source of danger of metastatic auto-infection or copious hemorrhages.

In my experience *patients with right-sided lesions of this type are more likely to recover than those with left-sided lesions.* In the former the constitutional symptoms, especially dyspnea, tachycardia, etc., may

improve or disappear after the formation of a chronic cavity and the disappearance of the pyrexia. Even dextrocardia may be well borne. But in left-sided lesions the heart is pulled over to the left and upward, and the patient remains with tachycardia and is distressingly short-winded. Pneumothorax is more likely to occur in the left pleural cavity.

The rational explanation for the mildness of right-sided lesions as compared with those in the left side is this: The left lung is smaller than the right and has but two lobes. The division of the lung into lobes retards the spread of the tuberculous process—the interlobar fissures, lined with double layers of serous membrane, act as barriers. In the right lung with three lobes there are two fissures, while there is only one in the left lung, and when this is passed, the entire lung is invaded. In addition, in extensive left-sided lesions, the diaphragm is drawn upward and with it the stomach, while the heart is pulled over to the left and upward; in some cases the apex beat may be found in the third interspace in the axillary line. The result is almost invariably disturbances in the circulation due to mechanical causes; the dyspnea is severe; more so than in dextrocardia found in right-sided lesions. Gastric symptoms, due to displacement of the stomach, are also very frequent in extensive lesions of the left lung. While I have seen many cases with cavities in the right lung and dextrocardia recover, I have seen but few with large excavations in the left lung do well. They may last for many years, but they are always unable to do anything because of severe dyspnea, cyanosis, etc.

In chronic cases in which the formation of a cavity is slow, the prognosis is rather good. In fact, cavity formation, as we have already shown, is a sign of immunity. Those with little or no resistance succumb before there is an opportunity for cavity formation.

These cavities are surrounded by dense fibrous capsules which limit their progress or extension, and they may be harmless for long periods of years. Communicating with bronchi which permit the expulsion of the morbid secretions forming on the ulcerated wall, they often pursue an apyretic course. Some even have smooth and glittering walls without any lymph spaces, and the toxic products within them cannot be absorbed. We meet with cases in which even the tubercle bacilli disappear from the sputum and the prognosis is the same as in bronchiectasis.

There are many of this class of patients who, despite having more or less extensive excavations, live for many years without pronounced inconvenience; in fact, some consider themselves fairly healthy and attend to their callings, or even to manual labor. Their main trouble consists in a proclivity to "catch cold," and only on such occasions do they call on their physicians for relief.

Generally speaking, tuberculous cavities are indications of chronicity of the tuberculous process in the lung, showing that the resisting forces are active and as such are of better prognostic augury than many active incipient cases with pronounced constitutional symptoms.

Patients are to be told that the "holes" in their lungs *per se* are not so dangerous as they believe. That fever, anorexia, etc., are more dangerous. They may live and can be active with cavities for many years.

Special Tests.—Various attempts have been made to find tests of the severity of phthisis by examination of the blood, urine, etc. We have already seen that Arneth's blood picture is not so reliable as some would lead us to believe (see p. 283). Ehrlich's diazo-reaction was at one time considered reliable in indicating the severity of phthisis. But it appears that it is positive in cases which are otherwise indicating their progressive tendencies. In incipient cases it is, as a rule, negative, but I have met with cases in which it was positive, yet the case went on to uneventful recovery. It appears that at present very few place great reliance on this test.

Moritz Weisz[1] found that urochromogen is the principal substance which causes the diazo-reaction, and suggested that his test is superior to the latter. I used Weisz's urochromogen test and found it superior to the diazo-reaction in indicating the prognosis of active phthisis. It is thus performed: Into each of two small test-tubes are put 8 cc of urine and 2 cc of distilled water are added; now, to one tube which is to be tested for urochromogen, 3 drops of 1 to 1000 solution of potassium permanganate are added, the tube is shaken thoroughly and compared with the control tube. The appearance of the faintest yellow color shows the presence of urochromogen and is easily detected by comparing with the control tube, to which no potassium permanganate is added. The test is read positive, however, only when the solution stays clear.

In this country Heflebower,[2] and J. Metzger and S. H. Watson[3] have reported that this test is a reliable guide in estimating the activity of the tuberculous process and gives indication as to prognosis. I find that it is positive during acute exacerbations of the disease and is usually negative in incipient, or even in quiescent, cases. In acute progressive cases it is found positive, and it becomes more and more intense with the extension of the disease. It is negative in most favorable cases.

The complement-fixation test, which has of late been used in the diagnosis of tuberculosis with doubtful results (see p. 392), has been found by some authors to have some prognostic value. Debains and Jupille[4] report that in active incipient and hopeful cases of phthisis the reaction is usually positive, while in advanced cases with pronounced emaciation the reaction is often feeble or altogether negative. They try to explain these phenomena on the assumption that in progressive and advanced phthisis the antibodies in the serum have already

[1] München. med. Wchnschr., 1911, **58**, 1348.
[2] Am. Jour. Med. Sci., 1912, **143**, 221.
[3] Jour. Am. Med. Assn., 1914, **62**, 1886.
[4] Compt. rend. Soc. de biol., 1914, **76**, 199.

been bound or neutralized by the substances produced by the tubercle bacilli. They also found that in experimental tuberculosis in rabbits complement-fixation activity goes hand-in-hand with the resistance of the animal. On the other hand, in tuberculous pleurisy with effusion negative reaction was mostly found, and this form of the disease cannot be considered as of especially unfavorable prognosis. In fact, we have shown that the outlook in pleurisy is rather bright. Most of the work along these lines was done by Besredka,[1] who reported that the reaction is uniformly positive in early cases of phthisis; in moderately advanced cases it is positive in the majority. With the advance of the disease the reaction becomes feeble, and finally in the terminal stages of phthisis it becomes negative. With Manoukhine he regards a negative reaction in advanced phthisis as a sign of approaching death.

From the results obtained by H. R. Miller in my wards at the Montefiore Hospital, the complement-fixation test showed no indications that it may be utilized for prognostic purposes. It has been found positive in active, as well as in quiescent, or healed cases, and as often negative in cases in which the contrary might have been expected.

Influence of Economic Conditions of the Patients on the Prognosis.— The occurrence of phthisis is in itself an indication of poverty. To be sure, we meet with numerous rich consumptives, but economic prosperity is not always an indication of rational life, proper food, regular hours, avoidance of physical and mental overexertion, etc. But in a given case of phthisis the prognosis is often influenced more by the social and economic condition of the patient than by any other single factor. After all, *phthisis is the most expensive of diseases* because it disables the patient for a long period of time and requires costly treatment, including nourishment, a favorable home, etc.

The patients who can afford to bear the expense are more likely to recover than those who cannot. The artisan often has a family depending on him for support, and he is likely to keep at work while sick, till the disease has progressed to a stage where he can do no more, and drops from sheer exhaustion. It is in these cases that the institutions, as well as the social service of modern enlightened communities, do considerable to improve the prognosis of phthisis. But it must always be borne in mind that these agencies can do much better than merely give advice about the dangers of living with tuberculous persons, and distribute scare head literature and sputum cups. If they do only this, the prognosis is often aggravated because the patient is, at times, treated like a pariah by his relatives and friends who are frightened by the numerous "visitors," the social workers, nurses, physicians, and others. I have seen families broken up in this manner; families in which there were no infants, and there was no reason to fear dissemination of the disease. But what is of most importance, the patient, deprived of the comfort of a good home, becomes despondent and the lesion progresses more quickly than it would otherwise.

[1] Ann. de l'Inst. Pasteur, 1914, **28**, 569; Compt. rend. Soc. de biol., 1914, **86**, 197.

Prognosis in Arrested Disease.—We have seen that only lesions of abortive tuberculosis are completely healed by cicatrization and calcification. But this form of the disease is not recognized, as a rule, during its activity and the prognosis is good at all events. It is different with chronic phthisis which has lasted for some time and finally there is an abatement in the constitutional symptoms and the patient is considered cured.

Cure by *restitutio ad integrum* is out of the question in these cases. The cicatrized and calcified foci usually contain virulent tubercle bacilli which may at any time become active again, flaring up the lesion or causing metastatic auto-infection. Experience has taught that in the vast majority of cases these patients attain but "quiescence," and the term "arrested disease," which has recently been substituted for the term "cured," which was formerly in vogue, is proper. The patient is justified in asking for an opinion whether this arrested condition is likely to be lasting, or whether he will sooner or later suffer from a recrudescence of the symptoms of phthisis, a relapse which is, in fact, an acute or subacute exacerbation. In other words, is the arrest of the disease an indication of a more or less permanent freedom from tuberculous sickness or is it merely a long remission in the progress of the disease?

These problems can be solved, in many cases, by a consideration of the physical signs found in the chest, but with greater certainty when the constitutional symptoms are considered.

Physical exploration of the chest discloses usually signs of cicatrization of the involved lung tissue, pleural adhesions, evidence of fibrosis, while the rest of the lung may show indications of emphysema. Adventitious sounds are usually, though not invariably, absent; the case is "dry." Exquisite amphoric breath sounds may be heard over the site of cavities, combined with amphoric whispered voice, but no rales. In others, the site of the lesion is only discovered by the dulness on percussion, and feeble breath sounds and sibilations are found over a circumscribed area of the chest, usually the upper part of one side. In many there are found signs of displacement of the mediastinum. But we have already emphasized the fact that the physical signs elicited on the chest are of but little value prognostically. The writer is under the impression that *a patient showing a well-defined line of demarcation between the normal lung and the affected part has a better prognosis than one showing a gradual change from normal to pathological lung tissue.* But to this there are many exceptions.

The problems, "Will the quiescence last?" and "Is the patient in danger of a relapse of the disease?" can best be answered by a careful consideration of the constitutional symptoms. In general terms it may be stated that the patient is in danger of two accidents: (1) pulmonary hemorrhage; and (2) reactivation of the disease.

Pulmonary hemorrhage cannot be foreseen in these cases, nor can it be prevented. It may occur when the patient is in excellent condi-

tion. When not copious, it merely frightens him, but even brisk and copious hemorrhages are well borne by 98 per cent of patients; in fact, they feel better in many cases after recovery from the bleeding, and quickly recuperate. Some have one such large hemorrhage a few years after recovery from phthisis and feel well for many years thereafter, or even for the rest of their natural lives. But in about 2 per cent of these bleeders the hemorrhages prove fatal. As was already stated, these hemorrhages cannot be foreseen nor prevented. Those suffering from "recurrent hemoptysis" hardly ever perish because of the bleeding. The danger is a brisk hemorrhage occurring suddenly in one who may not have bled before.

An exception is, however, to be made in the case of streaky sputum. In many patients with well-healed lesions in the lungs, minute hemorrhages occur, especially after slight exertion, or acute non-specific infections of the upper respiratory tract, etc. So long as there is no fever, severe cough, etc., this is to be considered capillary hemorrhage, due to ruptures of minute bloodvessels in the sclerosed pulmonary tissue. These slight attacks of hemoptysis are a good sign of healing, and should not alarm the patient. It is different with copious attacks of hemoptysis of which we spoke above. They are liable to threaten life in rare instances.

Healing of the tuberculous process in the lung frequently leaves the patient with certain annoying symptoms for an indefinite time. Many have pains in the chest, which may be aggravated during meteorological changes. This is particularly observed in patients who have pleural adhesions. In some the pain is paroxysmal, coming on without any known provocative cause, lasting for several days, and disappearing. No lasting improvement can be attained by therapeutic intervention. But the patient may be assured that these pains are no indication of a recurrence of the tuberculous process, so long as there is no elevation in the temperature or an acceleration in the pulse rate.

The constitutional symptoms are better guides in prognosis as to the chances of a lasting quiescent period. Most of these patients with arrested phthisis remain emaciated, anemic, with wasted muscles, often presenting a cadaverous appearance. Despite this, many of them are very active at their avocations and, in fact, they display energy and perseverance which is surprising when considered in connection with their physical decrepitude. Some are rather well nourished despite the fact that physical exploration shows a lesion of various degrees of activity, from cicatrization to excavation. In my experience, patients apparently well nourished, with quiescent or arrested lesions of this class are not as a rule doing as well as those of the lean type, despite their well-nourished bodies. We should not allow ourselves to be deceived in attempting a forecast by the amount of fat the patient has, by the fresh and browned skin which is often merely a superficial mask of improvement, while the interior of the organism is vitally undermined.

The prognosis in these two classes of patients can only be determined with some degree of certainty by an analysis of the following conditions: *If the improvement has been attained through careful treatment in a favorable environment, the test is whether the patient remains in good condition for some time after returning to his old environment without suffering a relapse of the constitutional symptoms.* The test, in other words, is duration; improvement counts if it lasts without special treatment.

So long as there is but little cough, or none at all, no fever, no tachycardia, dyspnea, chills, sweats, etc., the prognosis is good, no matter what physical exploration discloses. Continuous freedom from these symptoms for several months is an indication of arrest, even if tubercle bacilli are found in the sputum, while in those in whom arrest has just been attained, the prognosis is uncertain until time has shown that there is no tendency to recrudescence. The prognosis is even better in those who, despite resumption of their previous occupation or taking up a new one, and living a rational, though not an exceptionally careful life, still keep in good condition. On the other hand, *in those who purchased quiescence or arrest of the disease by special treatment, rest, and extreme care, the prognosis is less favorable, unless resumption of ordinary activities of life proves that recrudescence does not occur.*

In short, the prognosis of quiescent and arrested disease can only be made by a careful observation, for several months, noting the effects of resumption of activities of life on the condition of the patient.

CHAPTER XXXII.

THE MEDICO-LEGAL AND INSURANCE ASPECTS OF TUBERCULOSIS.

LASTING for many years in most cases, tuberculosis disables the patient more or less during the period of illness. If he has been insured in one or more of the corporations, or fraternal organizations which guarantee a certain sum per week or month for loss of working or business time owing to disability due to sickness, the physician must certify as to the ability of his patient to attend to his vocation. With the modern methods of workmen's insurance, it may be alleged that the victim of tuberculosis has acquired the disease as a result of the nature of his work, or because of lack of precautions to prevent the spread and development of tuberculous disease, and thus the question of compensation arises. Numerous men, having served in the army during the war, are now being cared for by the Government because of their disability due to tuberculosis acquired during or soon after military service. In these cases, examining physicians have to determine the liability of the Government, the extent of the disability, etc. Tuberculosis begins in most cases insidiously and there is time for the patient, knowing his condition or not, to apply for life insurance, and it is the province of the physician acting as examiner for the insurance corporation, to detect the disease, even though the applicant gives him no clue as to constitutional symptoms. Very frequently claims for damages are brought in courts because of actual or alleged traumatic origin of pulmonary tuberculosis. Expert medical testimony is then sought to advise the court whether, in the given case, the tuberculous process in the lungs may be charged to the injury. These and kindred subjects will be discussed in this chapter.

Disability Resulting from Tuberculosis.—The acute and progressive forms of the disease clearly disable the patients and no question can arise as to the patient's ability to work or attend to his business. The fever, cough, emaciation, weakness, etc., are self-evident and, as a rule, the insurance company or fraternal organization will not contest the claim for payment of the benefit promised in the contract.

It is different with the chronic forms of tuberculosis. These, as has been shown throughout the clinical parts of this book, are characterized by an undulating course, with exacerbations and remissions in the activity of the disease. The problem then arises as to the ability of the patient to work and the liability of the company. It seems that health insurance companies, once they are satisfied that the diagnosis of pulmonary tuberculosis is correct, especially when tubercle bacilli

have been demonstrated in the sputum, raise no more the question as to the disability and its extent and, as a rule, keep on paying the benefits. The writer knows of a case in which the insurance company settled with a policyholder for a flat sum believing that with tubercle bacilli in the sputum recovery is unlikely. The policyholder, however, soon became aware of his ability to work and has been doing so for two years.

In attempting to decide as to ability to work of tuberculous policyholders the physician should be guided mainly by the constitutional symptoms, while signs elicited while exploring the chest should be given a subordinate position. In some cases the general appearance of the patient is sufficient to decide. Those who are evidently emaciated and weakly, with a hectic flush, etc., can be put down as disabled. The examination of the chest is then made for the purpose of ascertaining the cause of the disability, and the extent of the lesion in the lung. In most cases a detailed examination is necessary before we can arrive at a decision as to the extent of the lesion and the outlook for recovery. It may be stated that a patient with a tuberculous lesion in the lung who has fever above 99.5° F. by mouth, or 100° F. by rectum, is not fit for work of a remunerative kind. There are many exceptions, to be sure, and they have been discussed in Chapter IX, but when an insurance policy is involved, disability must be considered complete in these cases for the time being. Likewise, those with tachycardia, with a pulse rate of 90 and more per minute, even when afebrile, must be considered disabled. In many instances functional tests must be applied, such as exercise of a more or less vigorous nature before the real character of the disability is ascertained. If, after performing some task, such as walking one or two miles, or working at the policyholder's occupation for an hour or two, the pulse, temperature and respiration remain normal, the individual may be considered fit for work; at most, payment for partial disability may be allowed. Of course, when these persons with dormant, or quiescent tuberculosis begin to work, it is imperative that they continue under medical supervision for several months, reëxamined at first once a week for two or three months, then monthly, and when it is found that work for a year has not deleteriously influenced the weight, temperature and pulse rate, they may completely be discharged with the admonition that with the first appearance of symptoms they must report for reëxamination.

It will be noted that cough and expectoration have not been mentioned among the constitutional symptoms which are invariably disabling. Many tuberculous individuals who have completely recovered remain with more or less cough; some even expectorate mucous or mucopurulent material for years, or indefinitely, but they remain strong, and well able to pursue their vocations. Even the finding of tubercle bacilli in the sputum is not inconsistent with ability to gainful employment in some cases. Likewise, the signs elicited on physical

exploration of the chest are not reliable criteria *per se* as to disability. In many instances it will be noted that, despite the unmistakable signs of extensive involvement of the lung, even of excavations, the general condition of the patient suffers but little, if at all; this may be seen in patients with cavities which cannot be considered "dry." On the other hand, in many cases in which the physical signs indicate but slight or even doubtful lesions, the constitutional symptoms are so pronounced as to completely disable the patient. The same may be said about roentgenographic findings. They merely indicate anatomical changes. But whether the changes disclosed on the plate are active, or disabling, cannot be determined with exactitude in every case merely by an examination of the roentgenogram. In fact, healed, cicatrized lesions at times cast a more pronounced shadow than some active lesions. Here, again, only constitutional symptoms decide.

Hemoptysis, especially the form in which the sputum is merely streaked with blood, cannot be considered as completely disabling, when there are no other symptoms of active disease. We have shown that recent investigations have demonstrated that exertion is hardly ever responsible for bleeding from the lungs (see p. 242); we have also seen that in some instances in which the lesion is, for practical purposes, cicatrized, the patients keep on expectorating blood-streaked sputum at irregular intervals for many years without reactivation of the process in the lungs (see p. 632). In some cases with cavities more or less profuse bleeding may occur, and soon after the hemorrhage ceases, the patient feels well, or even better than before the accident. Such hemorrhages should be judged by the after-effects. If, after the bleeding has ceased, the temperature and pulse rate are normal, and the patient regains the loss of blood and weight he sustained during the hemorrhage, he is often fit for resuming his occupation. Overexertion and excitement are hardly ever a cause of copious, or fatal hemorrhages, though they may be coincidental, and for this reason they have been exploited by writers of fiction, and on the stage.

The dangers of reactivation of healed tuberculous lesions as a result of moderate exertion have been greatly exaggerated by physicians and patients especially in health resorts and sanatoriums. No claim can be made to the satisfaction of careful clinical observers that patients with inactive or healed lesions, who keep at perfect rest have no relapses indefinitely. One has to scrutinize carefully the histories of patients who had been at rest in institutions for many months, or years, and it will be observed that, despite the rigid rest cure, reactivation occurs now and then without any discoverable causes. The psychic factor must be borne in mind, especially when dealing with patients who have been thoroughly institutionalized, in either public or private and expensive sanatoriums. They often become psychasthenic, introspective, and lazy; they have acquired the "thermometer habit," and cannot be weaned from it; they suffer from "hemophobia" (see p. 248), and fear the least exertion. They are difficult to manage and fraternal

organizations have them on their hands indefinitely. Recently the Government has had quite some trouble with this sort of cases among ex-service men.

Of course, in many of these healed or arrested cases of tuberculosis complete rehabilitation is not attained, and allowance for partial disability is to be considered. This is seen in patients in whom the lesion has healed excellently, but sclerosis of the affected lung tissue, as well as pleural adhesions, cardiac displacements, etc., leave him short-winded, with cyanosis of the lips and fingers, pains in the chest, etc. They may last for many years, perhaps their natural duration of life, and when they finally succumb, death is due to some other disease. But they lack endurance while they live. They cannot be considered rehabilitated, even though the lung lesion is practically healed, perhaps even better than in others in whom the disability is slight, though the lesion is apparently active or at most quiescent. It is in these cases in which the judgment of a physician with extensive experience with tuberculous patients is important when the problems of sickness, insurance or the liability of the Government to ex-service men are considered. Each case should be judged by itself. But in this, individualization, the reaction of the body to work, functional tests in other words, should be the basis for an opinion, and here again the temperature, pulse-rate, etc., are the criteria.

Nature of the Occupation.—Insurance companies assure an income for loss of business time, or inability to pursue a gainful occupation, because of sickness. Some occupations are completely barred to those who have had tuberculous disease. Among these are such as require strong muscular exertion, dusty trades, or those necessitating exposure to the vicissitudes of the weather. It is, in fact much safer for these persons to work indoors, provided the workshop, factory or mill is well ventilated and lighted. Guided by the principles laid down in Chapter XXXV, the medical examiner for insurance companies and fraternal organizations may be in a position to determine the hazards of working at most occupations.

Dangers of Infection.—The dangers of infection of fellow-workmen, which at times have been invoked by those who claim compensation, are to be disregarded. The number of "carriers" of tubercle bacilli is so large that the task of separating them from non-carriers cannot be attempted, and could not be accomplished even if attempted, with all the powers of the Government. Moreover, we have shown that there is no danger for adults because they have practically all been infected during childhood, and that this early infection gives them a certain degree of immunity against renewed and exogenous infection with tubercle bacilli (see Chapter V). Of course, in cities in which the health officers prohibit patients with a history of tuberculous disease from working at certain occupations, mainly involving the handling of food and food products, a change of occupation may be necessary in some instances.

TRAUMATIC TUBERCULOSIS.

Injury as a Cause of Extrathoracic Tuberculosis.—That traumatism may determine the localization of extrathoracic tuberculous lesions—of bones, joints, glands and meninges—is a well-known and accepted clinical fact, supported by animal experimentation. A considerable amount of information on this subject may be found in the works of R. Stern,[1] A. Ascarelli,[2] F. Parkes-Weber,[3] Léon Giroux,[4] H. Grau,[5] and many others. There appears, however, a considerable difference of opinion as to the frequency of traumatic tuberculosis in daily medical practice. Giroux collected the following figures as to the frequency of traumatism as a cause of surgical tuberculosis: Jeannel found that 5 per cent of cases are post-traumatic; Wilner, 6 to 7 per cent; Pietrzikowski, 8 per cent; Lemgey, 8.81 per cent; Estor, 9.5 per cent; Hahn, 31 per cent (of hip-joint disease); Honsele, 44 per cent; König, 20 per cent; Voss, 21 per cent; Horzetzky, 44 per cent (tuberculosis of the vertebræ); Taylor, 52 per cent; and finally Bauer, almost 100 per cent.

The differences in the percentages, running from 5 to nearly 100 per cent, show that some writers have greatly exaggerated the role of traumatism in the etiology of surgical tuberculosis. Indeed, many recent writers are inclined to the opinion that only in exceptional cases is traumatism responsible for the development of tuberculosis of bones and joints. Inasmuch as these forms of tuberculosis occur mainly in children, it is clear that an inquiry will nearly always elicit a history of an injury, usually a slight one, because but few children pass through a day or a week without hurting themselves in some manner. Moreover, the symptoms coming on insidiously, the child may only begin to complain after some injury and thus mislead the parents and the surgeon into the belief that the tuberculous lesion was the result of an injury.

As traumatic tuberculosis may be considered lesions resulting from inoculation of the tuberculous virus into the skin, such as the pathologist's and the butcher's warts, as well as the rare cases of infections when an open wound is inoculated accidentally while breaking a sputum receptacle, etc. But, as was already shown, these are extremely rare cases. An injury inflicted with a blunt instrument and causing merely a contusion, cannot produce tuberculous disease, unless there are tubercle bacilli somewhere in the body. However, in the light of our present knowledge of phthisiogenesis, it is clear that many, if not most, persons harbor some latent or healed tuberculous foci with virulent tubercle bacilli, which an injury may reawaken into activity. Reducing the vitality and resisting powers of the tissues at the injured

[1] Ueber traumatische Entstehung innerer Krankheiten, Jena, 1910.
[2] Policlinico, 1907, 14, 1025.
[3] Traumatic Pneumonia and Traumatic Tuberculosis, London, 1916.
[4] La tuberculose pleuro-pulmonaire traumatique, Paris, 1915.
[5] Handbuch d. Tuberkulose, 5, p. 69.

point, hematogenous or lymphogenous metastatic localization of tuberculosis is favored.

Traumatic Pulmonary Tuberculosis.—It has been known for a long time that injuries to the chest may favor the development of pulmonary tuberculosis. Goethe attributed his pulmonary hemorrhage at eighteen years of age to overexertion and a fall from a horse. But very few authentic cases were observed during the past century; Grasser could find reports of only 50 cases before 1903. In the Prussian army it was observed that among 6924 cases of phthisis, 95 began after injuries, and of these 79 had sustained contusions of the chest. This would indicate that it is more frequent than was formerly appreciated.

It is clear that primary tuberculosis cannot be caused by an injury to the chest. The only explanation we can offer for tuberculosis following traumatism is that after a contusion of the lung, the pulmonary tissue is devitalized so that tubercle bacilli brought in with the inhaled air will find a suitable soil for growth. Theoretically this mode of origin is possible. But to prove such a case it is necessary that a new lesion should be found during an autopsy, while careful search should fail to reveal old tuberculous foci anywhere else in the body. According to Grau such a case has, so far, not been reported. But many cases have been observed in which latent tuberculous lesions in the lungs, or other organs, have been stirred into activity soon after injuries. Inasmuch as we may confidently state that practically everybody has been infected with tubercle bacilli at some period of his or her life, but that in the vast majority of people this infection proves harmless, it is clear that, for medico-legal purposes, active tuberculosis following an injury should be considered a valid cause for claims for damages when an attempt is made to determine the responsibility.

In persons known to be tuberculous the disease may be aggravated by an injury, as the writer has seen in many instances, and lead a mild case to a rapidly fatal termination. Especially is this true when hemoptysis is caused by the injury. While in the usual hemorrhage from the tuberculous lung recovery is, as a rule, rapid, the blood soon regenerates, and the patient often feels even better than before the bleeding occurred, it is different when an injury is the cause of the hemorrhage. In such cases we usually deal with a contusion of pulmonary tissue, which prepares the soil suitable for the growth of tubercle bacilli, involving extension of the morbid process in the lung. Traumatism may also produce pleurisy, usually dry, but occasionally with an effusion. Pneumothorax is another possible result of an injury to the chest. In non-tuberculous pneumothorax the rent in the visceral pleura heals more or less quickly and the air is absorbed. Effusions in these cases are usually serous or serosanguineous, and are also absorbed. Even pyopneumothorax in a non-tuberculous individual is usually cured by surgical intervention. But in those with preexisting tuberculous lesions in the lungs or pleura, active or dormant,

the course of tuberculous pneumothorax, hydrothorax, pyopneumothorax, etc., may be followed. The prognosis in these cases is very grave (see p. 533). When the ribs are fractured, which is not uncommon in these cases, interstitial emphysema, subcutaneous and mediastinal, may occur and mediastinal emphysema may prove fatal in rare instances.

Intensity of the Injury.—The intensity of the injury should not be taken as a measure of the probability of its relation to phthisis subsequently developed, as has been pointed out by many writers. After violent injuries to bones and joints, especially those resulting from fractures, tuberculous osteomyelitis is hardly ever observed. During the World War very few gunshot wounds of bones and joints, and especially of the chest, were followed by tuberculous lesions in these tissues. On the other hand, slight injuries to bones have frequently been seen to precede tuberculous osteomyelitis. In the same manner the writer has seen in several cases a slight injury to the chest flaring up a latent, or quiescent tuberculous process in the lung. In persons known to be healthy, this is not uncommon. John B. Hawes[1] points out that after the autumn football season some players develop consumption as a result of injuries received on the football field. The special diet usually prescribed by the trainer, as well as the excessive exertion for months during the training period, undoubtedly reduce the resisting powers of even gridiron heroes. Parkes-Weber says: "Muscular men, such as athletes, runners, football players, boxers, and persons who live healthy, open-air lives and who, owing to their splendid physique, might be supposed to be specially resistant toward tuberculosis (*but are, it must be remembered, peculiarly liable to contusional injuries and strains*), sometimes surprise every one by more or less suddenly falling victims to tuberculous affections, especially of the lungs. When one hears of vigorous men, living 'healthful' open-air lives, becoming victims of pulmonary or other kinds of tuberculosis, one should always remember the possibility of some sort of traumatism having played a part in the etiology. . . . On some parts of the continent bargemen, who push their chests against poles, etc., are, I believe, said to be peculiarly liable to pulmonary tuberculosis." In this country tuberculosis often terminates the career of well-known athletic men.

Clinical Aspects of Traumatic Tuberculosis of the Lungs.—The site of the lesion in the lung provoked by an injury is not necessarily at the point affected by the blow. Many have observed lesions by *contrecoup*. In rare cases acute general miliary tuberculosis results from breaking up of a latent lesion and flooding tubercle bacilli into the blood stream. Hemoptysis immediately following the injury is not absolutely essential to establish the relationship between the injury and the disease, for laceration of the lung may occur without

[1] Boston Med. and Surg. Jour., 1913, **168**, 83.

causing hemorrhage. When hemoptysis occurs, as it does in many cases, the quantity of blood expectorated is no criterion of the size of the torn vessel, or the extent of the area of lung tissue contused. Nor must there remain any external marks on the chest wall because an injury may lacerate the lung without leaving any external traces.

The appearance of clinical symptoms of phthisis may be delayed for some time. In cases of quiescent lesions which are activated as a result of traumatism, the aggravation in the condition of the patient, and the extension of the process, may appear soon after the accident, and hemoptysis may appear even immediately. In many cases the bleeding is, however, delayed several hours or days, which is to be expected considering the pathology of hemoptysis. In apparently healthy persons the symptoms of phthisis may appear many months, or even years, later. Hawes reports several cases in which phthisis developed from two to ten years after the injury. The writer has given testimony in similar cases.

The appearance of demonstrable tubercle bacilli in the sputum may be delayed for weeks and months, and this does not militate against the traumatic origin of the disease. We know that in many cases of spontaneous phthisis bacilli are found only months, or even years, after the onset of the disease.

It takes about eight weeks for a tubercle to develop, and one tubercle is by far not enough to give symptoms or signs by which it can be recognized by the patient or the physician. In fact, when a few days after an injury signs of phthisis are found, especially tubercle bacilli in the sputum, we may conclude that we are dealing with a preëxisting lung disease which, at most, has been aggravated by the accident. But in cases in which the symptoms, such as fever, emaciation, cough, expectoration, etc., make their appearance three to six months after the injury, in a person known to have been well before the accident, and the physical signs of the local lesion appear even later, it is obvious that there was a causative relation between the injury and the disease. German authorities have limited the time for the appearance of symptoms after an injury to six months, although there are undoubtedly exceptions which must be judged on individual merits. It appears that in our army we recognize responsibility for several years, inasmuch as it appears to be the policy of the Government to care for tuberculous veterans for years after discharge from the service.

In many cases symptoms of acute pleurisy make their appearance within a few days—chilly sensations, fever, pain in the chest, dyspnea, etc. Usually these disappear within a few days, and are later followed by symptoms of phthisis. In some instances there occurs a pleural effusion which runs its course in the same manner as the average case of this type not due to traumatism, unless it is due to a rent in the visceral pleura with resulting hydro- or pyopneumothorax. With fractures of ribs, subcutaneous and mediastinal emphysema may occur in addition to pneumothorax. Hemoptysis has not been frequent in

cases observed by the writer, excepting in those who had suffered from pronounced phthisis for some time before the accident and the traumatism was the exciting cause of the hemorrhage. In such cases the amount of blood lost may be considerable. In most instances the hemoptysis is rather slight, a few mouthfuls, or merely streaky sputum is brought up.

Physical exploration of the chest may disclose the lesion right under the site of the injury on the chest, but at times it is found far away from it, even in the opposite lung, by *contrecoup*, as was already stated. This is an important point in cases in which responsibility for the disease must be established. In several cases of *basal phthisis*, the lesion being located in one of the lower lobes of the lung, I found it due to injury. In one instance it was a kick with the hoof of a horse; in another, a fall on the side sustaining a contusion of the chest; in still another the rebound of the crank while starting an automobile injured the chest.

The course of the disease may be acute, subacute, or chronic, and any of the clinical forms of tuberculosis may be observed. In fact, there is hardly any difference to be discerned in this regard between traumatic and spontaneous phthisis. The outcome depends mainly on the resisting powers of the patient.

The writer has observed several cases of acute miliary tuberculosis, and acute pneumonic phthisis following injuries. In such cases it was clear that the injury was inflicted on some part of the body in which there was a quiescent, or dormant tuberculous process. In one instance the patient was struck with a bottle over the chest. He immediately had a copious pulmonary hemorrhage, and within twenty-four hours the temperature rose to 104° F. and kept at this level for about six weeks, accompanied by symptoms and signs of acute pneumonic phthisis, terminating fatally. F. Parkes-Weber reports a case in which an injury set free a caseous focus in the epididymis, producing fatal phthisis. In fact, injuries to tuberculous testicles are very frequently followed by acute miliary tuberculosis with meningeal complications, as has already been shown (see p. 588).

Surgical Operations.—The development of acute and progressive tuberculosis after surgical operations may be classed with traumatic tuberculosis. This is mostly seen after operations for some chronic tuberculous disease of a joint, bone or gland which is followed by symptoms of acute miliary tuberculosis and ends fatally. The writer has seen cases of this sort develop after bloodless operations and manipulation of joints, and Parkes-Weber,[1] Urban,[2] Orth,[3] and others report similar cases. Among these may also be included the development of tuberculosis after childbirth and abortions. Parkes-Weber also mentions massage as a possible etiological factor in reactivating

[1] Loc. cit.
[2] München. med. Wchnschr., 1899, **14**, 346.
[3] Berl. klin. Wchnschr., 1914, **41**, 246.

dormant tuberculous processes and producing acute progressive disease.

Expert Testimony in Cases of Traumatic Tuberculosis.—Traumatic tuberculosis has of late been a cause for claims for damages and the physician testifying in such cases, irrespective whether he qualifies as an expert or not, must be prepared to discuss the following points: (1) Whether there is tuberculous disease in an active stage; (2) if so, has it been caused by the alleged injury; (3) in cases in which a previously existing tuberculous lesion has been aggravated by an injury, it is important to bring out the manner in which such aggravation occurs; (4) the effects of the tuberculous process on the working capacity of the plaintiff; (5) the outlook for a permanent cure; (6) the effects of the disease on the plaintiff's social life.

The first four points have been discussed in detail in the preceding pages. But it is important, when testifying for the plaintiff, to point out that even if curable, tuberculosis leaves a stigma on an individual living in a community in which the dangers of infection have been emphasized, and even exaggerated. Though it is entirely unjustified, people shun one with tuberculous disease, present or past, will not rent him a room in their homes; that it may interfere with his or her plans for marriage; that he is really a menace to infants whom he may infect by even slight contact, etc. It is also to be brought out that even after the disease is arrested, there is a possibility, in many cases, a probability, of a relapse which will disable him, or at least interfere with his working capacity.

MEDICAL EXAMINATION FOR LIFE INSURANCE.

It appears from statistics published by various life insurance companies that the losses sustained by these corporations paying death claims during the first few years of insurance are immense. Charles Lyman Greene[1] quotes a detailed report of one of the largest and most carefully conducted American Companies, which represents an analysis of 46,525 deaths constituting the total mortality from 1847 to 1898 inclusive. Of these deaths no fewer than 5585, or about 12 per cent of the total mortality, were charged to tuberculosis; 3307 deaths occurring in policyholders during the age period of twenty to forty-five, while in the age period of twenty-five to thirty they represented 32 per cent of the total mortality. No fewer than 594, or 18 per cent of the former group, died during the first two years of insurance, 1769, or 53.5 per cent, during the first five years, and in the latter age period (between twenty-five and thirty) 10 per cent of deaths from tuberculosis occurred during the first year of insurance, and 30 per cent during the first two years.

Bearing in mind that the insured are a select class, having been

[1] Medical Examination for Life Insurance, Philadelphia, 1905, p. 198.

examined medically before acceptance, and all who show any of the stigma of disease are supposed to have been eliminated, the following figures are illuminating.[1] They represent a comparison between the mortality rates from pulmonary tuberculosis in twenty-four Registration States and the experience of the Industrial Department of the Metropolitan Life Insurance Company.

	Twenty-four Registration States.	Metropolitan Life Insurance Company, Industrial.
1914	123	185
1915	123	180
1916	121	173
1917	125	172
1918	130	171
1919	109	142

It is clear that despite the attempts on the part of the medical departments of the insurance companies to weed out all who have tuberculous lesions, and also that a large number of deaths due to tuberculosis in insured persons are recorded as due to other causes, a fact well known to medical men, the loss sustained by these corporations, and by fraternal organizations, is excessive. There is no doubt that careful medical examination, by physicians qualified to do it, would materially reduce the number of deaths due to tuberculosis during the first three years of insurance and save millions to these companies and the policyholders. Of course, it cannot be expected that all be weeded out by medical examinations, because it is evident that policyholders are just as liable to develop tuberculosis as others. Careful medical examinations could also prevent the rejection of applicants for policies for alleged tuberculosis, or so-called predisposition to the disease, without justification, as is observed daily in medical practice. In such cases an injustice is done to both, the insurance companies and the applicants, for obvious reasons.

Elimination of Tuberculous Risks.—Obviously those who suffer from the active and progressive forms of the disease are rejected. In the advanced stages of the chronic forms of the disease this is clearly an easy matter for the medical examiner. In most cases the hectic flush, the emaciation, tachycardia, dyspnea, etc., are sufficient. In some instances, however, external appearances are deceptive; patients with large cavities in the lungs, with thick and adherent pleuræ, etc., may present a healthy countenance, and perhaps even be of normal weight, or over. In incipient cases, and they are liable to succumb sooner on the average than the just mentioned class, external appearances are of little, if any, value in estimating an insurance risk.

In attempting to eliminate tuberculous individuals the examiner may be guided by the following points.

The Family History.—It appears that the value of the family history in the diagnosis of tuberculosis has been overestimated. While the

[1] Public Health Reports, 1921, **36**, 1178.

death rates of infants descended from tuberculous stock is very much higher than of others, adults of this class have not been shown to develop phthisis to a degree as to bar them from insurance. Familial tuberculosis affects mainly infants; the susceptible are weeded out during the first few years of life. Statistics carefully collated do not prove conclusively that descendants of tuberculous parents who survive to adolescence are all doomed to end in phthisis. There is good evidence to the effect that when they do develop tuberculosis, the disease is likely to run an exceedingly chronic course, tending to recovery (see p. 621). Insurance companies have, however, found by experience that a family history of tuberculosis in the ascendency results in an excess of death claims. Rusher and Kenchington[1] investigated this problem among a group of 80,000 policyholders with a family history of tuberculosis, the total experience comprising 622,462 policy years, and an average observation period for each policyholder of nearly eight years. They found that the mortality experienced in the early years of insurance was heavy, especially for young ages at entry. After the first five years of assurance the excess mortality is less marked, especially in the case of endowment policies, in which it comes well within normal limits. The latter fact suggests the importance of "selection" by the insured; of the "bad life" more or less consciously choosing a whole-life policy rather than an endowment. For these reasons, in addition to very careful examination, those with a family history of tuberculosis should be given rated policies when found otherwise acceptable.

Past Diseases.—Signs of tuberculous disease during childhood, which are mainly seen as scars remaining after diseases of the glands, bones and joints, should not in themselves be taken as grounds for rejection. There are but few persons who had not been infected with tubercle. which has left traces in the intrathoracic glands, pleura and lungs. But these pathological changes are not perceptible to the average examiner. If those with scars on the neck are to be rejected efforts should be made to discover all who have sclerosed and calcified glands at the hilus of the lung for rejection, which is obviously not feasible nor rational. We have repeatedly shown that these glandular and osseous stigmata of past tuberculous disease are no signs of excessive predisposition to tuberculous disease in the adult; in fact, there is ample evidence of the effect that it is a sign of resistance higher than the average. The fact that it is exceedingly rare to find scars on the necks of patients with active tuberculous disease of the lungs, attests to this contention (see p. 586). Here again, when tuberculosis does develop in these persons, which is exceedingly rare, it usually runs a very chronic course, tending to recovery. However, these individuals are apt to develop acute miliary tuberculosis with implication of the meninges. Fifty per cent of cases of tuberculous meningitis in adults

[1] Jour. Institute of Actuaries, 1913; quoted from Tubercle, 1920, **1**, 161.

have had extrathoracic tuberculous disease, especially of the genito-urinary tract, active, quiescent or healed. For this reason this class should not be accepted without reservation. Applicants with scars of the kind just mentioned, showing no signs of tuberculous disease of the lungs, who are well nourished and appear generally healthy, may be accepted on rated, or perhaps endowment policies. Straight whole-life policies should not be given to them. This suggestion is supported by the statistical evidence gathered by the American Medico-Actuarial Investigation of 1914 in which it is shown that in insured persons who gave a history of an attack of tuberculosis of bones (hip, etc.), there were 44 deaths as against an expected number of 23.14; the ratio being thus 190 per cent of the expected mortality. In insured persons whose history indicated an attack of bone tuberculosis more than ten years before the application, the ratio was only 120 per cent of the expected mortality.

Pleurisy is most important in this connection. We have shown that an enormous proportion of those who recover from pleurisy develop pulmonary tuberculosis sooner or later (see p. 512). The statistics gathered by the Medico-Actuarial Investigation of 1914 showed that in insured persons giving a history of an attack of pleurisy within five years prior to the application, the death rates were about three times the normal; five to ten years prior to application, about twice the normal; and when the history indicated an attack over ten years before application for insurance, the death rate was about normal. For this reason, all applicants with a history of pleurisy should be rejected, at least within ten years following the attack of pleurisy. We have shown that most cases of tuberculosis following pleurisy occur within ten years after the acute disease, and that more than three-fifths of patients pass through life without developing phthisis. Moreover, a distinction is to be made between so-called primary, or "idiopathic" pleurisy, which is with but few exceptions tuberculous, and those accompanying pneumonia, especially influenzal pneumonia, which are not tuberculous. But the medical examiner who sees the applicant years after the attack of this disease can hardly make this fine distinction. Hence, applicants who have had pleurisy, but have been well for ten or more years, and on careful examination of the chest no signs of any active tuberculous process can be discovered, may be considered for rated policies. A history of pleurisy during childhood, and of empyema, is not to be considered as a load, or as predisposing to tuberculosis.

Of other diseases, it is important to mention that influenza, which was formerly considered as predisposing to tuberculosis, has been found to have no influence on the subsequent development of this disease (see p. 128). If the patient has recovered and shows no signs of pulmonary changes in the lungs or pleura, the later evolution of tuberculous disease will depend on factors other than the attack of influenza. Likewise, persons "subject to colds," if these "colds" are not in themselves indications of active tuberculosis of the lungs, which can be

discovered by an examination of the chest, are no more predisposed to tuberculosis than others. Applicants "subject to colds" should therefore be carefully examined; postponed in doubtful cases. If, on several examinations, spread over a period of six months or a year, the cough and expectoration are found to be undoubtedly due to rhinopharyngeal changes, while in the lungs no pathological changes can be detected, tuberculosis should be disregarded within the same limits as in the average individual.

A history of having been in a sanatorium for tuberculous patients should excite great diligence while examining the applicant, and caution before the issuance of a policy is recommended. It must, however, be borne in mind that sanatorium residence does not invariably prove that the patient has been tuberculous, or was so even at the time he lived at the institution. A large number of "suspects" are admitted, kept for months in sanatoriums, and discharged as "non-tuberculous," or as "cured" of a disease which they never had. Many are discharged as "suspects." We have already shown that 20 to 50 per cent of sanatorium patients are sputum negative (see p. 584), and hardly 15 per cent of persistently sputum negative cases are sick with phthisis. The writer has seen many cases that were rejected by life insurance examiners merely because they had been in sanatoriums though they never had any conclusive symptoms or signs of tuberculosis. A careful inquiry should be made in every sputum negative case as to the symptomatology before, and during residence at the institution, the course of the disease for which treatment was applied, and especially as to the sputum findings. If these point to a non-tuberculous condition, and there are no signs of any lung lesion, past or present, particularly if the applicant is examined more than five years after his residence at the sanatorium, he may be considered for a policy. Several postponements may be necessary in some cases. Outright rejection is unjust in many cases.

On the other hand, those who had undoubted symptoms of tuberculosis while at the sanatorium should be rejected. Elsewhere in this book are given statistical investigations by Elderton and Perry, Noel Bardswell, and others, on the mortality of patients after sanatorium treatment (see p. 726); they show that they are not good insurance risks.

A history of *hemoptysis*, even without a distinct history of tuberculous disease, while not invariably proving that the subject is likely to develop active phthisis, yet is sufficiently serious to excite caution on the part of the examiner. The Medico-Actuarial Mortality Investigation found that policyholders with such a history gave the following mortality:

A. One attack within five years of application	151
B. One attack between five and ten years of application	131
C. One or more attacks, the last more than ten years prior to application	102

In group A the death rates from tuberculosis were fully five times the normal. In group C they were nearly twice the normal. It is thus clear that a history of hemoptysis is a distinct load, and justifies rejection even if the physical signs elicited on the chest are not conclusive of an active lesion.

History of Exposure to Infection.—This may be disregarded because there is hardly a resident in a large city, or a worker in any industrial or mercantile establishment brought into contact with many people, who has not met with tubercle bacilli "carriers." If exposure to infection were a load, physicians would surely have a higher mortality rate from tuberculosis. It appears that when infected those who had not come into contact with tubercle bacilli carriers, are likely to contract acute and progressive tuberculous disease. Primitive people, brought into civilized countries, would make very poor risks for life insurance companies because of their liability to contract tuberculosis in its acute forms. Yet they were born and raised in an environment free from tubercle bacilli. Intimate exposure to infection has not been found effective in increasing the rate of morbidity and mortality from tuberculosis in adults. As was shown at length (see p. 149) conjugal tuberculosis is very rare. Römer mentions that life insurance companies in Germany, basing their action on statistical experience, do not reject persons because of a history of exposure to infection, or who live with tuberculous consorts. George Florschütz[1] in his work on insurance selection, says that "in medical selection one must certainly consider the risk of infection when it is so evident as in conjugal intercourse, but in general, as far as life insurance is concerned, one may regard tuberculous infection as purely a matter of chance." He brings statistics showing that "of 1428 deaths from tuberculosis, there were but 11 in which the husband and wife of the deceased were tuberculous." We have also shown that healthy workers in sanatoriums and hospitals for consumptives are no more liable to tuberculous disease than others (see p. 147). The average adult living with a tuberculous individual in one house is hardly as much exposed to infection as are physicians, nurses, and orderlies in hospitals and sanatoriums.

General Appearance.—In its relation to tuberculosis the weight of the applicant for a policy should be considered to a certain extent. Fat individuals only rarely develop phthisis. On the other hand, "fat phthisis," though rare, does occur and, irrespective of the weight of the applicant, a careful examination of the chest is to be made. Persons under weight are to be carefully examined for physical signs of lung disease because phthisis is the most common cause of emaciation. Bisbee[2] found that those above the standard weight gave 5.59 per cent of consumptive deaths; those with standard weight, 25.91 per cent; those below standard weight, 42.51 per cent. These figures

[1] Medical Record, 1915, **87**, 957.

[2] Quoted from Greene, loc. cit., p. 365.

were based on 3548 cases. However, it is well known that many of the slim and wiry individuals live long. An applicant for a policy who is underweight, who shows no signs of present or past tuberculous changes in the lungs, and is incidentally free from sugar in the urine, or indications of some other cachectic disease, may be passed in accordance with the rules of the company. In most cases in which loss of weight is a recent acquirement, and these are the ones that must be guarded against, there is a flabby skin, and caution is indicated.

The following external signs should direct that attention of the medical examiner for vigilance while examining the chest: A hectic flush; skin eruptions of the type of pityriasis versicolor, acnitis, folliclis (see p. 269), etc.; enlarged veins on the chest, especially unilaterally; clubbed fingers, inequality of the pupils, atrophy of the muscles of respiration, excavations above and below the clavicles and supraspinous fossæ. Wherever any of these signs are noted on inspection in one who is underweight, the physical exploration of the chest must be thorough and systematic.

Physical Exploration of the Chest.—No proper examination of the chest can be made unless the applicant's clothing is entirely removed to the waist. Experts in physical diagnosis, and they are as rare among medical insurance examiners as among the profession in general, will frequently fail to detect a gross tuberculous lesion in any stage of the disease when the examination is made while the chest is covered. Incipient and limited lesions can hardly thus be detected and persons with incipient lesions only too often apply for life insurance. Raising the shirt is insufficient; it must be completely removed. As far as the writer's observations have taught him, this is frequently not done.

The appearance of the chest will give ample clues, if properly observed and interpreted. The flat chest in itself is no indication of active tuberculosis, as was already indicated (see p. 302), and it is decidedly wrong to refuse insurance to one merely because he has a narrow, flat and long chest, when no symptoms and signs of any tuberculous changes in the lungs can be elicited. To be sure, this form of chest is, in many cases, an indication of past tuberculosis, especially of the tracheobronchial glands. But we have shown that it points to healed tuberculous adenitis of the type most people pass through during childhood. These persons are not tuberculous, and there seems to be available evidence showing that they have been to a certain degree immunized against the graver and more acute forms of this disease. Acute progressive phthisis is most frequently seen in individuals with excellent and capacious chests, in athletes, pointing to lack of acquired immunizing forces which protect the average individual. On the other hand, local retractions of the chest wall, atrophy of the respiratory muscles, etc., should be given careful attention. Scars indicating operations for empyema should not be considered of importance in themselves as indicating tuberculous lung disease, present or potential, because empyema, in adults as well as in children, is not a predisposing factor

to tuberculosis. Likewise, deformities of the chest, the result of rickets during infancy, the rachitic chest, are no indications of an excessive predisposition to tuberculosis.

In looking for signs of tuberculosis most attention should be concentrated on the region of the lungs above the third rib, the apices. With but few exceptions, rales, when heard unilaterally in these regions are indicative of tuberculous disease. No applicant should be recommended for a policy in whom rales are heard over the upper third of the chest anteriorly or posteriorly. It is immaterial what kind of adventitious sounds are heard exclusively in these regions, be they dry or moist, consonating, whistling, sonorous or sibilant, if they are heard over the apex, especially unilaterally, they offer sufficient ground for rejection.

If tuberculosis is to be detected in its early stage, when applicants for insurance are apt to apply for admission, it is even more important to scrutinize carefully the breath sounds heard over the upper lobes of the lungs. We have already emphasized the fact that in really incipient lesions no rales may be heard at all but that in nearly all cases there are certain, though variable, changes in the character of the breath sounds (see p. 339). The following hints may prove of assistance in efforts to eliminate tuberculous risks: If the breath sounds are weak, feeble, or absent in one of the apices, there is ground for caution. Urging the applicant to cough may provoke rales, or crepitation, in the same area. In others, the expiratory murmur may be prolonged, or bronchial in character; in some bronchovesicular breath sounds are heard. If the resonance over the apex is impaired over the area with feeble breath sounds tuberculosis is to be thought of. It is well to bear in mind that these changes are usually heard in early cases over the "alarm zone" (see p. 379). Also that changes in breath sounds limited to a circumscribed area, are more important in this regard than when heard all over the chest.

It is, however, unfair to reject an applicant for insurance merely because of some slight changes in the breath sounds and resonance of the chest, even when found over the apices, especially the right apex. In many cases this is one of the anomalous physiological phenomena consistent with perfect health and longevity; or it may be an indication of some pathological process in the lung or pleura which has never given trouble and healed perfectly. In such cases it is urgent that certain other features of the applicant should be inquired into before a decision is arrived at.

Constitutional Symptoms.—The most important effect of active tuberculosis is the production of toxic symptoms. Without symptoms of toxemia active tuberculosis is rare. These can best be determined by a study of the constitutional symptoms of the case as outlined in other parts of this book. It is clear that the insurance examiner is not in a position to make a careful clinical study of the applicant. Moreover, at the time he submits to an examination the applicant may

be afebrile, though if carefully studied, it would be found that he runs one of the various types of fever already described as characteristic of tuberculosis in the incipient stage (see p. 217).

But there is one symptom of tuberculous toxemia which is only rarely lacking and cannot be concealed. Tachycardia is one of the cardinal symptoms of active tuberculosis; bradycardia is extremely rare; even a normal pulse rate is very uncommon. Hence, if there are some doubtful physical signs detected in the chest, and the pulse rate is rapid, the applicant is to be rejected. If nervous influences are suspected as the cause of acceleration of the pulse, postponement is in order. This is a point which, if followed, would prevent many medical examiners from passing tuberculous applicants for insurance; it would also prevent the rejection of healthy persons because of some slight alterations in the resonance and breath sounds of the chest. When the rate of the pulse is around normal, 70 or less per minute, and the applicant appears well nourished, slight alterations in the breath sounds may be disregarded. At any rate, the applicant may be postponed for three to six months, and if there are no changes in the physical signs, and especially if the nutrition of the patient is well maintained, he may be recommended for a policy, provided the examiner is satisfied that during the period of postponement the applicant continued at his occupation. If he has been away for a rest, this may be detected in many cases by noting the parts of the skin which are exposed to the action of the sun rays, the hands, shoulders, face, etc. As a rule, a vacation leaves unmistakable traces on these parts.

Following the above diagnostic principles might save the insurance companies millions which are annually paid out for deaths which occur during the first two or three years of insurance. It would also prevent the rejection of many applicants for insurance because of "tuberculosis" which is subsequently shown to have been non-existent.

CHAPTER XXXIII.

INDICATIONS FOR TREATMENT OF PHTHISIS.

THE indications for treatment in pulmonary tuberculosis appear at first sight to be simple and clearly defined. On the principle that the first thing to do is to remove the cause, it would seem that there are but two procedures to follow: To destroy the bacilli which have settled within the body; or to increase the resisting powers of the patient, and thus render the soil unsuitable for the growth of the invading virus. But in this case, the ideal, like other ideals, cannot be achieved in the average case, and the aim at curing the patient by the first of these procedures is not feasible at the present state of our knowledge.

We have no chemical remedy which will destroy the bacilli harbored within the body without simultaneously killing the patient. We have no drug which will render the tubercle bacilli harmless in the body, as quinin destroys the malarial parasite, or arsphenamine and mercury destroy the spirocheta in syphilis, leaving the patient in good shape. Even the so-called specific treatment—the various tuberculins, sera, and vaccines—which have been lauded for their alleged curative powers when properly administered, are not stated to have any known bactericidal action, nor are they known to hinder the proliferation of the bacilli within the body, or to immunize the tissues against the poisons engendered by these microörganisms through the production of antibodies, as is the case with antitoxins. Attempts at active and passive immunization have not met with notable success in tuberculosis.

The etiology of tuberculosis, however, teaches a lesson in rational therapeutics. The tubercle bacilli do not grow with equal facility in every individual; if they did, the number of human beings who succumb to this disease would be equivalent to the number that give positive reactions to tuberculin, indicating that they have been infected with tubercle bacilli—over 90 per cent of the adult population in large urban centers. We have seen that the bacilli can proliferate and produce their noxious effects only in persons who offer a favorable soil for their existence.

In what this favorable soil consists, we are not altogether clear. In the chapters on Phthisiogenesis we discussed it in detail, and it was evident that everything which undermines the general health of a person and reduces his vitality may prepare a favorable soil for the growth of tubercle bacilli within the body, and thus produce phthisis.

As a corollary we may argue that anything which will stimulate the vital defensive forces, which are more or less inherent in every individual, or which will improve the nutrition of the body may hinder the proliferation of the bacilli, and with the improvement in the general physical condition of the patient the local lesion may cicatrize, or the dissemination of the bacilli by metastasis may be prevented.

This is what modern phthisiotherapy is aiming at in handling each individual case of the disease. As has been pointed out by G. Schröder,[1] modern therapeutic tendencies, which are based on the achievements of immuniology, have not changed our methods of treatment of tuberculosis, especially phthisis. It is today, as it has been hitherto, based on the general principles of therapeutics, because phthisis *as a disease* cannot be considered an infectious disease *sui generis*. It can only originate in individuals with a certain constitutional susceptibility, which may be inherited or acquired.

Air, Food, and Rest.—The traditional therapeutic triad—air, food, and rest—has withstood the test of time, and is at present called into service more often than ever before in the treatment of phthisis. Indeed, like many other excellent therapeutic agents which have become standard, it is very often abused. Many patients know of it and quite often tell their doctor that they are aware of the fact that medicine is helpless and that air, food, and rest are all that they need. Curious to say, some physicians do not protest.

But this is all wrong. The medical man of today has many more resources in his attempts at curing phthisis and should not rely on the above-mentioned triad exclusively. Indeed, a physician who advises a patient to lead an open-air life in some region famous for its beneficial effects on this disease, and urges him to consume more and better nourishment than he has been in the habit of taking, and to stop all life activities, fulfills but part of his duty to his patient. There are many more therapeutic resources which hasten recovery, relieve the most annoying and painful symptoms of the disease, and go a long way toward prevention of complications, which cannot be met by the above-mentioned indications.

Effects of Polymorphism of the Disease on Therapeutic Indications.—Since the etiological unity of tuberculosis has been proved by the discovery of the tubercle bacillus, the profession has tacitly accepted that unity of origin invariably implies unity of effect, and the treatment of the disease was also unified. But this is an error. We have seen that the tubercle bacilli produce different lesions in different individuals, as regards the anatomical changes in the lung, the clinical phenomena, and the course and curability of the disease. Indeed, there are hardly two cases of phthisis which appear exactly alike on the autopsy table, and all the groupings into caseous, fibroid, cavitary, pneumonic, etc., are inadequate. This is especially true of the clinical

[1] Handbuch der Tuberkulose, 1914, **2**, 1.

manifestations of the disease; its polymorphism is noteworthy and important. To be sure, this is also true of other diseases, notably syphilis, yet the specific remedies in the latter answer most of the indications. *So long as we are not in possession of a specific remedy or tuberculosis, it will have to be treated symptomatically.*

Under the circumstances, to be effective, treatment must be applied in accordance with the clinical manifestations encountered, and to a certain extent with the clinical form of the disease. We have seen that each form pursues a course more or less different from all other forms. It would therefore be wrong to treat a patient with abortive tuberculosis in the same manner, and for the same length of time, as one with chronic progressive phthisis. Fibroid phthisis demands different treatment from chronic caseous phthisis; febrile cases cannot be treated like those which run an afebrile course. The various complications of the disease, like intestinal, laryngeal, and renal tuberculosis, demand special care which the general indications do not satisfy. Preëxisting disease, like syphilis, diabetes, cardiovascular and renal derangements, etc., alter the course of treatment appreciably. There are also differences in our methods of treatment when we care for a tuberculous child, as compared with those applied in adults; but in senile phthisis the indications are not the same as those in adolescents. The indications are even different in cases of young, single women, as compared with married or pregnant women, and during the menopause tuberculosis often demands special treatment.

It is thus obvious that a method of treatment which will suit all cases cannot be formulated. What may be efficacious in one may not be feasible in another, or even harmful in a third. *The treatment of phthisis must be individualized to suit the case; it must be elastic and adaptable to the polymorphous nature of the disease and to the various accidents and complications occurring during its course.*

Criteria of Efficacy of Treatment.—In judging the value of any method of treatment, we must bear in mind some points which are usually neglected while speaking of this subject. *The fact must not escape us that the vast majority of cases of tuberculosis manifest a strong tendency to recover under any method of treatment, or even spontaneously.* Impressed by the malignancy of the disease in many cases, we are apt to forget the large number of spontaneous recoveries, and when we meet with good results, we are likely to attribute them to the method of treatment pursued, forgetting that a large proportion of patients would have recovered without the treatment.

Discussing the clinical features of abortive tuberculosis, we have shown that this form of phthisis is very common and may not be recognized. When reading about a large proportion of recoveries in a sanatorium which admits only "incipient" cases, or of a drug which is alleged to cure at this stage a certain proportion of cases, etc., we must recall that among these "early" cases, there are a large number with a strong tendency to recovery under all circumstances. *To be of real*

value, a method of treatment must be effective in producing more recoveries than would be ordinarily anticipated.

Even in the forms of chronic phthisis which usually last for many months or years before terminating in recovery or death, the course is not always progressive, continuously advancing. This is evident from the large number of patients who give a history of hemoptysis, cough, fever, emaciation, pleurisy, etc., five, ten, or more years before the onset of the present illness, which was diagnosticated at the time as tuberculosis, but the patient did well. For long years he had been able to attend to his work, only being laid up now and then for a few days with an attack of "bronchitis," "grippe," etc., but this last attack has proved persistent. Now, if in this case a proper diagnosis had been made during any of the previous attacks, the prompt recovery would have been credited to the special treatment applied. In fact, many patients tell us that a certain prescription was very effective for years in relieving them promptly, but this time it has failed.

All properly investigated statistical examinations have shown conclusively that five years after the onset of active phthisis about 50 per cent of the patients are in good or fair physical condition and even able to make themselves useful at their respective occupations irrespective of what method of treatment was applied. The statistics of results obtained in sanatoriums show that patients discharged in the advanced stages of the disease are often found alive and active, five, ten, or even fifteen years later. A physician who keeps careful records and publishes a series of cases in which such results are shown can impress the profession that his method of treatment has done wonders. Yet it is just what should be expected under any harmless method.

A study of the literature on phthisiotherapy shows that nearly all authors, urging their methods, report certain and almost the same percentages of patients "cured," "disease arrested," "improved," "unimproved," and last, but always least, "dead." Practically all sanatoriums, whether located on high or low altitudes, at the seacoast or inland, in cold, warm, or moderate climates; irrespective of the special method of treatment pursued—indoors, outdoors, or in tents; no matter what the fad or hobby of the attending physician, be it dietetic, medicinal, or specific; they all give the same results if we should judge them by the percentages of reported cures, improvements and deaths as published in their annual reports.

During the first year or two after the introduction of new drugs or specifics, physicians report excellent results, as is seen from the literature on creosote and arsenic and their derivatives, ichthyol, cinnamic acid, iodin, tannin, succinimide of mercury, etc. They all cured a certain percentage, arrested the disease in a larger percentage and failed only in very acute, progressive, or far-advanced cases. Phthisiotherapy has thus been encumbered with an enormous number of medicaments which have been lauded by many competent and con-

scientious physicians at one time or another, and condemned with equal vigor by others. According to Rénon the popularity of each drug or method of treatment hardly exceeds three years.

These are, in fact, the reasons why so many new methods of treatment, drugs, specifics, climates, diets, etc., are annually announced as curative agents for tuberculosis. They all depend on the normal proportion of recoveries which occur under any method. That charming French writer, Louis Rénon,[1] says in this connection: "All new therapeutic methods of treatment of tuberculosis, so long as they are harmless, always give the same satisfactory results. This is an axiom which I should like to have printed with heavy type in all the new books on phthisiotherapy. It is an axiom which may be clinically translated into this simple statement: Hurry and take the treatment as long as it cures: if you wait you may be too late."

The reasons for these therapeutic illusions are found in the above-stated facts. The disease is acutely progressive in comparatively few cases. In these, all agree that their remedies are of no avail and they are not counted in the reported cases. In a large proportion there is a strong tendency to spontaneous cure, and they furnish the recoveries for the special climates, specific and empiric therapeutic agents, for the "milk cure," the "song cure," the "grape cure," etc. In the majority of cases of active phthisis the disease runs an undulating course, with more or less frequent exacerbations of acute or subacute symptoms, followed by remissions in the activity of the process. In some the acute exacerbations are very infrequent, long remissions are obtained, the patient feeling comparatively well for several months and the credit is given to the method of treatment.

Psychic Influences.—Persons under the influence of mild alcoholic intoxication are very susceptible to suggestion, and the consumptive who is under the influence of tuberculous toxemia is very vulnerable to auto and heterosuggestion, as was shown in Chapter XIII. Any new drug, especially when boosted in the newspapers, is apt to relieve him in a remarkable manner. We often meet with consumptives who keep on sinking while under the care of a physician, but for some reason are impelled to change their medical adviser and, though the latter makes no changes in the treatment, the patient begins to gain in health and general well-being. This is usually the result of a new, careful, and minute physical examination by some pedantic physician who subjects his patient to all the diagnostic procedures—inspection, palpation, percussion, and auscultation; "gives him the benefit of the latest of diagnostic aids," the *x*-rays, the cutaneous or subcutaneous tuberculin test, examines the sputum and urine in the presence of the patient, etc., and usually gives the same directions as those of the former physician, but more minutely; orders the patient to report frequently to see whether any changes are necessary. This is often

[1] Le traitement pratique de la tuberculose pulmonaire, Paris, 1908, p. 30.

the beginning of a most remarkable improvement in a case that has been going from bad to worse: The appetite returns, the cough ceases, the nightsweats disappear, etc., and he gains in weight and strength.

Suggestion by Tuberculin Treatment.—There are many phthisiotherapists, competent to give authoritative opinion, who are convinced that tuberculin, as generally administered in minute doses, acts more by suggestion than by specific action on the tuberculous process in the lung. We shall revert to this subject while speaking of specific treatment. But meanwhile we want to point out the powers of suggestion in specific treatment as shown in a drastic manner by Albert Mathieu and Dobrovici,[1] who announced to the tuberculous patients at the Andral Hospital in Paris that a new discovery had been made, a new serum had arrived for the cure of tuberculosis, and that shortly a sufficient quantity of the remedy would be available for those in need of it. The patients had to wait for some time, and when the serum arrived they all rejoiced. The new remedy consisted simply of physiological salt solution, but was given the pompous name *Antiphymose*. Certain patients were told that they were fit subjects for antiphymose, while others were denied the treatment on the plea that it would not do them any good. The selected patients were placed under careful observation and their histories were again recorded minutely, so that all felt that they had been seriously given the first opportunity to benefit by a great discovery. No change was made in the surroundings of the patients and the diet, but all other medication was discontinued.

The patients were greatly impressed by the new remedy and the favorable results exceeded all expectations. Within a couple of days there was noted an improvement in the appetite; those who had fever before showed a normal temperature, and the cough, expectoration and nightsweats were ameliorated; those who had hemorrhages ceased bleeding, and even the physical findings in the chest showed distinct signs of amelioration of the process. The gain in weight was remarkable, ranging from 1500 gms. to 2 and 3 kilos. As soon as the injections were discontinued all the old symptoms reappeared.

From personal experience[2] with the culture of turtle bacilli injected by Dr. F. F. Friedmann into patients under my care at the Montefiore Hospital in New York City, I can say that its effects were practically the same as those of Mathieu's antiphymose. The heightened susceptibility to suggestion of the average consumptive was here vividly illustrated. No one will deny that the vast majority of people, healthy and sick, are amenable to suggestion in various ways, but it must be acknowledged that a group of patients suffering from acute or subacute gout or rheumatism, heart disease in a state of decompensation, of nephritis complicated by dyspnea and dropsy, of ulcer of the stomach, of cancer, or of any other organic pathological entity,

[1] Bull. gén. de therapeut., 1908, **151**, 882.
[2] Fishberg: Interstate Med. Jour., 1914, **21**, 349.

would not be influenced to the same extent by suggestion as were the consumptives just mentioned.

It appears that consumptives in all stages of the disease are susceptible to psychotherapy. I have repeatedly observed marked improvement in the subjective symptoms of patients who were told by their physicians that nothing could be done for them because they are doomed, while the new physician, who was promptly called because of the extreme prostration of the patients, assured the unfortunate sufferers that there was no danger at all, and that only careful treatment was necessary to rehabilitate the lost health and strength, and afterward a short visit to the country would enhance the chances for ultimate recovery. I have seen improvement in a patient after three punctures were made in her chest with a view of inducing an artificial pneumothorax, but no nitrogen was introduced into the pleura because of adhesions. Yet the temperature, which had been quite above normal for weeks, promptly dropped to normal and the patient felt well. That tuberculous patients, as a rule, improve during the first few weeks or months in a new resort or institution is a well-known fact; and that it is usually not the superior climatic conditions or the different method of treatment that is efficacious in this respect is proved by their relapse into their former condition, or by the aggravation of their disease, after the novelty of the new surroundings begins to wear off. This is the main reason why climates "wear out."

Psychotherapy in Tuberculosis.—This heightened susceptibility of the tuberculous patients to suggestion is of immense value and assistance to the physician who is the fortunate possessor of a personality which stands him in good stead when handling difficult and intractable cases. But it is a double-edged sword. It also interferes in a large measure with the proper appreciation of the value of any therapeutic procedure, because the patients are apt to be impressed with any new remedy, especially if it has been puffed up by an enthusiastic physician, and promptly improve. But the improvement is only short-lived, and within a short time all the old symptoms return, as we have shown.

This psychic trait of the tuberculous is, however, of immense value in assisting physicians in their efforts to alleviate the more painful features of the disease, provided they know how to take advantage of it. Indeed, the success of many physicians in handling tuberculous patients depends on this point, and it is a fact that therapeutic nihilists fail, as a rule, to give relief to this class of patients. The detailed, often written, instructions given by physicians to their patients in sanatoriums, the minute doses of tuberculin administered, the vigilant anticipation of reactions, and the careful inquiry as to the effect on the constitutional symptoms, have all the elements of suggestive therapeutics. Without these details, institutional treatment of tuberculosis, especially in private and costly sanatoriums, would be a failure.

For these reasons medicinal treatment of tuberculosis has a place

in the therapeutics of tuberculosis. The materia medica is of assistance not only in alleviating certain annoying symptoms, as we will show later on, but rational medication also imbues the patient with the idea that something is being done for him during his long and trying disease. Medicinal preparations are palliative, to be sure, but they often carry the patient over an acute crisis with more or less comfort which could not be obtained otherwise, and they stimulate a hopeful outlook for an ultimate recovery.

The Indications for Treatment.—In the absence of specific remedies the therapeutic aims are to increase the natural forces of resistance of the tissues by constitutional treatment and by direct local treatment of the affected lung. The first indication is met by certain general therapeutic measures, the second by the induction of an artificial pneumothorax. In this book the treatment of phthisis is discussed with a view of methodically presenting the subject in the following order:

1. Prophylactic treatment.
2. General management of the case.
3. Dietetic management of the case.
4. Institutional treatment.
5. Climatic treatment.
6. Medicinal treatment.
7. Specific treatment.
8. Symptomatic treatment.
9. Local treatment.
10. Treatment of the various forms of tuberculosis.
11. Treatment of the complications.

CHAPTER XXXIV.

PROPHYLAXIS.

THE recent discoveries in the field of phthisiogenesis have shown that the prophylaxis of tuberculosis is much more complex than the simple formulæ or programs of antituberculosis societies would indicate. A considerable part of the sure preventives given in popular and technical literature have been shown to be inefficacious or superfluous by the newer teachings of the bacteriology, immunology, demography, and the clinical phenomena of this disease.

Modern prophylactic measures should differ in accordance with what we aim at attaining. If our aim is to prevent *infection* with tubercle bacilli, we must take different measures from those which are indicated when we aim at preventing *phthisis*, the disease caused by these microörganisms. In our attempts at preventing tuberculosis in children we must resort to other prophylactic methods than when we aim at preventing tuberculous disease in adults. In fact, measures which are likely to prove effective in infants are not indicated in older children, while in adults most of the measures which have been found effective in early life are futile, extravagant, and even harmful.

Prevention of Infection.—We have seen that the child is born free from tuberculosis, even if its parents are tuberculous at the time of conception or birth. We have also seen that during the first year of life some become infected and that the proportion showing signs of harboring tubercle bacilli in their bodies keeps on gradually increasing with advancing years so that at ten years the vast majority are infected, and that at the age of fourteen over 90 per cent react to tuberculin—an unmistakable sign of having been infected with tubercle bacilli.

We have also shown that during the first year of life infection, if it does occur, is likely to result in an acute or subacute disease which proves fatal in nearly all cases. On the other hand, after passing the age of infancy infection becomes less dangerous, only rarely causing death, though it is liable, when localizing itself in glands, bones, and joints, to cause prolonged sickness and end in disfigurement, if the patient survives.

Our main aim is therefore clear. The infant under two years of age must be protected against tuberculous infection at all costs. In families in which there is no tuberculous member this is a simple matter. Impressing the parents that infants acquire tuberculosis very readily, as easily as measles, scarlet fever, influenza, etc., and that a single exposure is liable to result in infection, they can, with reasonable and ordinary care, shelter their young offspring against the tubercle bacilli. Especially is this an easy matter with mothers

who suckle their babies, and do not give them any cows' milk, so that bovine infection is entirely excluded.

An infant is naturally not apt to come in contact with strangers unless those who care for it bring it in their proximity. Realizing that there are so many persons with open tuberculosis who are considered quite healthy, or who consider themselves healthy, "carriers" in the full sense of the word, it is obvious that in order to positively avoid infection at that age, infants must not be brought in contact with any one excepting the immediate family who are known to be free from the disease.

But it must be remembered that the immediate family includes the grandparents, and they are often suffering from latent tuberculosis. The impression is gaining ground of late that a large proportion of the chronic bronchitis, pulmonary emphysema, asthma, etc., in aged persons, is of a tuberculous character, as was already shown in the chapter on Phthisis in the Aged. The writer in attempting to trace the source of infection has often found that it was the coughing or expectorating grandfather or grandmother who was responsible for the disease in an infant.

Great care is to be exercised in selecting domestic servants for homes with infants. Especial care is to be taken with the nurse for an infant. She should be carefully examined by a physician, and reëxamined if she acquires a "cold" that lasts more than a week.

These simple measures suffice in homes in which there are no tuberculous inhabitants. No infant should be allowed to remain in a home in which a phthisical person resides. Even if the patient is one of the most scrupulous, and takes excellent care of his sputum, he should not live in the same home in which an infant is raised. This is a point which, in our efforts to prevent the dissemination of the disease, is often overlooked. Following up phthisical patients, the authorities usually state that a careful consumptive is harmless, so long as he takes care of his expectoration, and permit tuberculous persons to live in the same home with infants. But as a matter of fact the harmlessness of consumptives extends only to adults, and not because they are taking extreme care of their expectoration, but for other reasons which will be given later on in this chapter. As regards infants, no care, however conscientiously exercised, can surely prevent infection. And infection in infants is likely to prove deadly.

The indications are therefore clear. Either the phthisical person or the infant is to be removed. No compromise can be allowed in such cases.

No tuberculous mother is to be allowed to rear her young children, especially during infancy. It has been found that very few infants survive when suckled by a mother suffering from phthisis. The extensive statistics of Weinberg,[1] embracing 5000 families with 18,000

[1] Die Kinder der Tuberkulösen, Leipzic, 1913.

children, have shown that the nearer the birth of the children to the time of death of their tuberculous parents, the higher the mortality among them. Three-fourths of the children born during the last year of life of tuberculous mothers succumb; and 90 per cent of the children born during the last month of life of tuberculous mothers die. The investigations of the present writer[1] among children of tuberculous parentage in New York City have shown practically the same condition to prevail. In addition to the excessive mortality in general, 16 per cent of the deaths among children under six years of age were due to tuberculous meningitis, as against only 1.27 per cent among the general population of New York City.

The prophylactic value of separation of the infant from its tuberculous parents is well exemplified by experiences with tuberculous animals. Harlow Brooks[2] shows that in cattle the question of whether or not the offspring becomes tuberculous depends entirely upon exposure after birth. It has been conclusively shown that the calves are very rarely, if ever, infected before birth, but that the slightest carelessness in exposure of the newborn calves to infections leads to certain disaster. It has been found that tuberculous animals may be utilized for breeding purposes and that they may be crossed and inbred with entire disregard of the factor of tuberculosis and purely for the purpose of improving or maintaining the type, provided the calves are separated from the parents immediately after birth.

Similar measures have to be taken in cases of newborn infants of tuberculous parentage. If the mother is tuberculous the infant is to be removed immediately after delivery, and should not be allowed in her proximity during the first two years of life. If the father is phthisical, he should be removed from the home so long as there are infants under two years of age. In some cases the alternative of removing the infant may be more feasible. Bernheim induced three tuberculous mothers who had twins to separate with one child each, while retaining the others in their homes, though healthy wet-nurses were employed to suckle the babies. The three isolated children remained healthy, while the three which were raised at home succumbed to tuberculosis. Armand-Dellile studied a series of 787 children born or living in 175 families one or more members of which were tuberculous. Of these children 323 were placed in the country and all did well; 396 were not removed from their tuberculous environment, and of these 328 developed tuberculosis. Figures like these show how imperative it is to separate infants from their tuberculous parents more drastically than any other evidence.

Available evidence tends to show that the infant is not infected through ingestion of the milk from its tuberculous mother, but through the bacilli she eliminates while speaking or coughing. Human milk is only rarely found to contain tubercle bacilli, so long as there is no

[1] Archives of Pediatrics, 1914, **31**, 96, 197.
[2] Am. Jour. Med. Sci., 1914, **148**, 718.

tuberculous disease of the breasts. Stanley L. Wang[1] and Frederick Coonley examined the breast milk of 28 tuberculous women; specimens from 15 cases were injected intraperitoneally into guinea-pigs. In all cases the results were negative, no tuberculous changes being found in the animals at the autopsy; 450 microscopic examinations of specimens of milk were taken bi-weekly from the whole series of 28 cases. These were all negative, excepting 1, which was positive once, and 1 other specimen from the same case, which was suspicious once. A. B. Marfan[2] reports similar experiences. He says that tubercle bacilli have only exceptionally been found in human milk. A few experiments have produced tuberculosis in animals after injecting them with milk taken from the breasts of tuberculous women. There are but two authentic reports of infants being infected by the milk of their mothers. These were the cases of Demme and Roger and Garnier.

It is noteworthy that improvement in the sanitary and hygienic conditions, which are so effective in preventing phthisis in the adult, as will be shown later on, are not of any value in the case of infants. As has been pointed out by Römer, it was found that scrupulous attention to hygiene and sanitation of the stable, such as proper construction, ventilation, cleanliness, etc., hardly has any influence on the prevalence of tuberculosis in cattle, and that only strict isolation of the sick from the healthy animals is effective. Primary infection in infants appears to follow the same law: Exposure of an infant, even in an ideal home, may result in fatal tuberculosis, while life under adverse conditions will not produce tuberculous disease, unless there is a source of infection, which is usually the human consumptive and rarely milk derived from tuberculous cows. In the development of phthisis in adults hygienic and sanitary conditions play, however, a very important rôle.

The prevention of bovine tuberculosis is not to be neglected. When an infant must be hand fed, the milk should be carefully selected. In large cities the only drawback is the cost. Certified milk is everywhere available, but it is rather expensive and prohibitive for the vast majority of the population. For this reason all milk that is not derived from a source known to be safe is to be pasteurized or, better yet, sterilized. Pasteurization does not always destroy all tubercle bacilli, as was shown by Hess. On the other hand, an investigation by E. C. Fleischner and K. F. Meyer,[3] in San Francisco, showed that in certified milk bovine tubercle bacilli were not present in sufficient number to infect guinea-pigs. Certified milk is thus the safest for infants. However, the main problem is the human bacillus, as was already shown.

These simple measures are to be taken with a view of successfully preventing primary infection of infants under three years of age. They can be easily carried out by any family that has some degree

[1] Jour. Am. Med. Assn., 1917, **69**, 531.
[2] Le nourisson, 1916, **4**, 34.
[3] Am. Jour. Dis. Children, 1917, **14**, 157.

of economic independence. In families which are to some extent hampered because of economic stress, the State is to interfere. Health Boards, which are busy protecting adults against *infection* to which they are hardly susceptible, could perform really useful service if they concentrated more and more along these lines. The mortality during the tender age of infancy, which has hardly been influenced by the campaign against tuberculosis, would be reduced to a minimum. Moreover, massive infection, which is apparently responsible for phthisis in adults who have survived it during infancy, may thus be largely prevented.

Prophylaxis in Children Over Three Years of Age.—When the child begins to walk around and comes in contact with many people, prevention of infection is not simple. The parents, especially those who cannot afford a maid for each child—and they constitute the bulk of the population—lose control over their children, unless they are prepared to keep them altogether from contact with strangers, and this is not feasible for obvious reasons. Later when they go to school, they are bound to come in contact with other children and adults, and it is altogether impossible to prevent their meeting tuberculous individuals, no matter what the economic condition of the parents. It is thus clear that it is quite if not altogether impossible to prevent tuberculous infection among children over four or five years of age.

But, as was shown in Chapter XXIV, infection in children over four years of age is usually relatively harmless. Either no disease at all occurs or rarely tracheobronchial adenopathy results, which is serious only in exceedingly rare instances.

Available evidence tends to show that in infants infection is usually accomplished within the family—tuberculosis is exceedingly rare in infants who live in homes in which there is no phthisical member. When this is the case, we may trace the infection to someone living in the house as a lodger, or to some relative or friend who visits the home and comes in intimate contact with the infant, thus causing massive infection. "It is said that after two infant children of a royal house had died of miliary tuberculosis it was discovered that their old nurse had a tuberculous infection of the maxillary sinus with a fistulous opening into the mouth through which mucus laden with tubercle bacilli frequently passed." (Bushnell.[1]) With children of play and school age, the opportunities for intimate contact with adult strangers are scarce; they are not taken in the arms, not kissed indiscriminately, etc., and even when infection takes place, it is from another child, a playmate, etc., is slight, and not so massive as it is likely to be in infants, who are infected from adults.

There is abundant clinical evidence of the relative harmlessness of infection of children over four years of age. One has but to consult the mortality returns in any country to convince himself that between

[1] Epidemiology of Tuberculosis, New York, 1920, p. 177.

three and fifteen years of age the mortality rates from tuberculosis are comparatively low, despite the fact that over 90 per cent of the tuberculous infection of humanity takes place during this period of life. Comparing the results of infection during the first two years of life, and those taking place between four and fifteen years of age, the contrast is striking and convincing (see p. 453). Neither acute tuberculosis nor chronic phthisis of the adult is common in children of school age. Thus, among 925,000 children examined by the medical school inspectors in New York City during the school year September, 1914 to June, 1915, only 68 were found tuberculous.[1] When we bear in mind that each was examined by physicians and nurses once in six weeks on the average, and that a complete physical examination was made of all children three times during the course of the elementary school year, and that a cough noted by the teacher was sufficient to refer the child for examination, it is obvious that not many suffering from tuberculosis were overlooked.

Under the circumstances, we may conclude that no matter what the cause is, infection of children during school age is comparatively harmless, and that, inasmuch as experience has taught that everybody is bound to be infected with tubercle bacilli, the best that can happen is that infection should occur at the age period of four to fourteen years. The primary mild infection at that age, as we have shown above, practically vaccinates humanity against more severe infections in later years. Otherwise, all adults would be as susceptible to tuberculous disease as are guinea-pigs or the indigenous races of Central Africa.

Our efforts are therefore to be directed, next to the prevention of contact of infants with tuberculous persons, at the prevention of massive infection of children. This can be done within certain limits by preventing children from associating with individuals suffering from open tuberculosis. The danger lurks mostly in adults, because children expectorating tubercle bacilli are exceedingly rare.

Prevention of Reinfection.—It thus appears that the bacilli infecting children remain dormant within the body and cause no disease so long as there are no predisposing or exciting causes. We know that under certain circumstances these dormant bacilli activate and cause disease by metastatic auto-infection. This is mainly seen in cases in which, owing to defective nutrition, or some intercurrent disease, notably measles, whooping-cough, typhoid, etc., the resistance is reduced, and an exacerbation of the tuberculous process takes place. Moreover, it appears that the younger the child, the more is the anergy thus induced likely to be followed by active tuberculous disease. The indications are therefore clear—young children and infants are to be sheltered against the endemic diseases. Special care is to be exercised in this direction with children of tuberculous parentage, who have in

[1] Weekly Bulletin of the Department of Health, City of New York, 1915, **4**, 289.

all probability suffered from massive infection. This class of infants is to be scrupulously shielded against measles, whooping-cough, scarlet fever, diphtheria, etc. If these diseases are bound to attack them, it is best that it should occur after they have passed the fourth year of life.

During convalescence after any of these endemic diseases, the child is to be given special care with a view of prevention of metastatic auto-infection while the body is in a state of anergy; in other words, susceptible to tuberculous infection. This may be accomplished by either taking the child to the country, preferably the seashore, for a few weeks or months, till it has completely recuperated; or, when it must be kept at home, it should be given proper nourishment, and kept outdoors the greater part of the day, and it should sleep in a room with open windows.

Artificial Immunization.—We have emphasized that tuberculous infection confers a certain degree of immunity against reinfection with the same virus (see Chapter V). It is this immunity of the tuberculous to tuberculosis that has suggested attempts to procure artificial immunity by vaccination with living tubercle bacilli. The most important work along these lines was done by Behring, who vaccinated cattle with human bacilli to which they have shown a certain degree of resistance. Römer did the same with sheep, and more recently F. F. Friedmann announced that he has succeeded in immunizing children against tuberculous disease by inoculating them with an emulsion of living turtle bacilli.

So far the results of animal experimentation along these lines have not been encouraging. It appears from Behring's and Römer's prophylactic inoculations that immunity was not attained in all cases, and in the few that it was, the resistance to infection was rather short-lived, lasting but a few months. Friedmann's claim that tuberculous disease is prevented by his turtle bacilli vaccine has been found unfounded. It is clear that the vaccinated children will have to be watched for at least thirty years before agreeing that they are immune to pulmonary tuberculosis. During childhood this disease is quite rare (see p. 453).

More recently, Calmette and Guerin[1] reported remarkable results attained by prophylactic inoculation of cattle with attenuated bovine bacilli. The fatty capsule which envelops the tubercle bacillus has been found to consist of an alcohol which is not unlike cholesterol. Bile salts are capable of dissolving, or at least holding in colloidal suspension, certain lipoids as cholesterol. Inasmuch as the difficulty encountered in attempts at destroying tubercle bacilli is due to their fatty capsules, bile is thought to be capable of rendering this capsule vulnerable. On this principle Calmette believes he attained attenuation of the tubercle bacilli by cultivating them for twelve years, through seventy

[1] Annales de l'Inst. Pasteur, Paris, 1920, **34**, 553; L'infection Bacillaire et la Tuberculose, Paris, 1920, p. 577.

transfers on glycerolated potato treated with bile. However, the few animals (cattle) which Calmette reports as having been immunized by the vaccine of these attenuated bacilli, are not sufficient encouragement to proceed with similar vaccination of human beings.

In general it may be stated that prophylactic vaccination has not as yet given encouraging results.

Prophylaxis in Adults.—Prophylaxis in adults is no more a problem of infection. It may be taken for granted that everyone who has passed through the first fifteen years of life, especially in a city, has been infected with tubercle bacilli. The fact that he shows no symptoms and signs of disease is no proof that he has escaped infection, as was already shown. In adults, the problem is the prevention of disease, of phthisis. I believe that a considerable portion of the inefficacy of the campaign against tuberculosis is due to the lack of appreciation of this distinction between infection and disease.

This fact is based on the newer investigations in phthisiogenesis, which have conclusively proved two points:

1. That chronic phthisis in the adult, of the type that creates most of the tuberculosis problem, never occurs immediately after a primary infection; if disease occurs at all soon after a primary infection, it is of the acute types of tuberculosis of the lungs or of other organs. Indeed, when disease follows immediately after a primary infection of an adult it is almost invariably deadly, as is seen in tuberculosis of primitive peoples who had not been exposed to infection during childhood.

2. Infection with tubercle bacilli, whether it causes disease or not, renders the body immune against further and renewed exogenic infection with the same virus. Inasmuch as nearly all adults have been infected with tubercle bacilli during their childhood, they are immune against reinfection with bacilli which may be eliminated by tuberculous persons. The phthisical manifestations in adults are attributed to infection during childhood, just as the tertiary manifestations of syphilis are late results of the original infection years ago, though the body is immune against renewed exogenic infection with the same virus.

If this were not a fact, practically all the workers in hospitals for consumptives would succumb to the disease: all consorts of tuberculous persons would acquire the disease. One has to consider that of women married to, and living with, husbands suffering from active syphilis, hardly any escape infection. But we see thousands of tuberculous persons living with consorts, having children with them, yet the unaffected consorts remain in good health, as we have already shown in detail (see p. 149).

It is therefore a vain effort to follow up tuberculous persons, push them from pillar to post, interfere with their employment, as has been done in many cases, with a view of preventing infection of fellow-workmen. If these individuals cannot infect their husbands or wives,

as the case may be, despite the intimate contact, they are surely not a menace to their fellow-workmen.

This fact is now beginning to be recognized by those who are well-informed about the recent progress in our knowledge of phthisiogenesis. There has been manifesting itself a reaction against the absurd and cruel phthisiophobia which has been rampant for about twenty-five years. Baldwin[1] says: "Adults are very little endangered by close contact with open tuberculosis, and not at all in ordinary association. . . . It is time for a reaction against the extreme ideas of infection now prevailing. There has been too much read into the popular literature by health boards and lectures that has no sound basis in facts and it needs to be dropped out and revised."

We have shown that while the mortality from tuberculosis has decreased during the past few generations, infection of human beings has gone on to a degree as could never have been higher. There are sagacious physicians who believe that it is fortunate for humanity that infection has not completely been conquered. "We have done our best during the last quarter century to diminish tuberculous infection," says Bushnell,[2] "and something has been accomplished, no doubt, in lessening the size and frequency of infecting doses. Fortunately as yet we have not succeeded in diminishing by one iota the morbidity of the disease. Not that such diminution, and that to the point of extinction, is not desirable, but we are not yet ready for it. For as soon as we diminish the morbidity the danger arises that the opportunities for tuberculization will likewise diminish, and after a deceptive lull cases of acutely fatal tuberculosis will begin to take the place of the former more benign types of the disease."

Prevention of Phthisis.—It appears that in the eager chase after the bacteria, which could never be entirely destroyed, we have forgotten that only a small portion of those infected develop phthisis, while the rest are apparently benefited by the infection. Some recent writers have not hesitated to apply the term *benevolent infection* to those who have been fortunate in acquiring tuberculosis during later childhood and have thus been immunized against primary infection after fifteen years of age, when the disease produced by a primary infection is apt to run an acute and fatal course. Otherwise, we would all succumb to the acute and fatal forms of tuberculosis.

Phthisis is a disease occurring in persons who have been infected with tubercle bacilli many years before the outbreak of the disease. It is due to reinfection. But available evidence appears to point in the direction that the reinfection occurs from within, that it is metastatic; the bacteria which have remained dormant for years are slowly or suddenly reawakened into activity, and they produce new lesions; and that exogenous reinfection is exceedingly rare, if at all possible.

We know that certain conditions favor a reduction in the normal

[1] Johns Hopkins Hosp. Bull., 1913, **24**, 220.
[2] Loc. cit., p. 183.

resisting powers of the body and permit the proliferation of the dormant bacilli. Among these, inferior sanitary, hygienic, and economic conditions stand out preëminently. We have seen that the rates of wages, the number of rooms in which a family lives, the character of the work pursued by an individual, etc., have a strong influence in the direction of enhancing or preventing the evolution of phthisis. For this reason, the philanthropic agencies may do more toward the prevention of phthisis by concentrating their attention on improvements along economic lines of reform than by sending agents to tell adults that it is dangerous to remain in the proximity of a consumptive. Labor unions do better by exacting higher wages and shorter hours than driving unfortunate phthisical persons from their places of employment, as is being done of late in New York City.

Phthisiophobia.—Phthisis is undoubtedly an exacerbation of dormant tuberculous processes in the lungs; its entire clinical course is undulating, with periods of quiescence interrupted by periods of activity. These acute and subacute exacerbations may be prevented by careful attention to the general health of any individual who shows the least tendency to phthisical disease. Such individuals should not be hounded, refused employment, etc. They are to be helped along in the direction of securing easy work during the quiescent periods, so that they may be self-supporting and self-respecting. The words of an intelligent and observing consumptive on this subject are to be borne in mind by social workers, who of late seem to know more of the etiology and prevention of tuberculosis than those who have made a special study of the subject. Says the American historian, William Garrot Brown, in his *Confessions of a Consumptive:*

"The public depends for protection from such danger as our continued existence involves, not on its own exertions but on ours. To render that protection we must burden ourselves with both expense and trouble. We must incessantly take, for the sake of the public, precautions which are disagreeable and costly; and meanwhile a great part of the public is, by its attitude toward us, steadily tempting us, and even sometimes fairly compelling us, if we would live to discontinue these precautions and go on as if there were nothing the matter with us. The folly and stupidity of this attitude it is impossible to overstate. It is of itself by far the chief cause and source of the persistence of this scourge.

"Known and recognized and decently entreated, we are not dangerous. Shunned and proscribed and forced to concealments we *are* dangerous. Victims ourselves of this same régime of ignorant and self-deceiving inhumanity, we are called on every hour of our lives for a magnanimous consideration of others. Society can hardly find it surprising or a grievance if our human nature should sometimes weaken under the strain of the incessant provocation it endures from this strange working of human nature in general. Why should we alone be expected to be guiltless, always to our own cost and sacrifice, of

that very form of man's inhumanity to man from which we ourselves are suffering more than anybody else? Yet I can honestly attest that the vast majority of us are guiltless of any merely resentful offense; that, as a rule, when we fail to protect the public it is only because the public compels us to disregard its interest, its safety. This is what I earnestly entreat the public, for its own sake, candidly to consider.

"Candidly means fully. If the public is to be safe from us, if the public is to continue to have our protection from that against which it failed to protect us, then the public must make it possible for us to get—it must certainly cease to make it impossible for the mass of us to get anything except by subterfuge—what we must have to live. We are neither criminals nor mendicants. We do not ask favors, we merely revolt against a mean and stupid oppression. We revolt against ignorance and against a lie. The public would get rid of us, and thereby makes us inescapable. It would pretend, and would have us pretend, that we are nowhere. It thereby insures that we shall be everywhere. It proscribes us and thereby admits us."

If the average consumptive was not shunned by adults; if he was permitted to work unmolested after he is cured or the disease is arrested, or quiescent, allowing him to earn his livelihood, a considerable part of the economic stress caused by this disease would be done away with. If the tuberculous individual is told that he is only a menace to infants, less dangerous to children, and not at all dangerous to adults, he will surely take all precautions against infecting those who may be harmed by it.

But at present the State, municipal, philanthropic and social agencies that send out representatives telling those who live with consumptives that the patients must be shunned, and incidentally conveying the information that a careful patient, *i. e.*, one who takes care of his sputum, is not at all dangerous, even to infants. Some patients in New York City are actually dreading lest their names will be reported to the authorities, and they will be pestered by those well-meaning nurses, physicians, social workers, etc. Instead of telling the patient that he is only a menace to infants, and that he must keep away from them, they often visit his place of employment and the result is that the unfortunate patient is soon without a job and starving.

The results of these methods of phthisiophobia are seen in the fact that the number of infants which succumb to tuberculosis has not decreased even in Germany where antituberculosis agencies have been most active; that the number of persons infected with tubercle bacilli has not decreased is clear when we consider that over 90 per cent of humanity react to tuberculin.

I do not want to be understood as speaking unfavorably of all prophylactic measures against tuberculous infection of adults. There are many, especially among the richer classes in cities, and in suburban and rural districts, who have escaped infection during childhood, and they should be protected. It is, in fact, well known that tuberculosis when occurring in these classes is often of an acute type, just as it is

in the indigenous races of Central Africa or in the Esquimaux. They should be protected against the sputum indiscriminately expectorated by consumptives, and against droplet infection when coming in contact with persons suffering from active phthisis. But with the city-bred people, especially those who have survived in the congested parts of cities or the slums, there is hardly any danger that adults will be infected with tubercle bacilli. They have been infected during childhood; vaccinated and immunized against additional infection. But it is just among these that the strong efforts are made to prevent exposure of adults to infection. The irony is that their infants are usually neglected by the social forces working in the antituberculosis campaign.

Just as cattle breeders have found that the control of tuberculosis is mainly a matter of prevention of infection of newborn calves, and that adult cattle may be disregarded, so must we act with humans. To prevent infection, newborn infants must be protected while children over ten and adults need no special measures, especially those who have been raised in cities.

Disposal of the Sputum.—In our attempts at preventing infection, the disposal of the sputum expectorated by phthisical patients is more important than any other prophylactic measure. The pathogenic bacilli are distributed in a virulent form only from one animal body to another. Exceptionally, the source of the bacilli is a domestic animal, mainly milk from tuberculous cows, but in the vast majority of cases the source of infection is sputum expectorated by phthisical patients.

For this reason the rigorous laws prohibiting indiscriminate expectoration which enlightened communities have inaugurated are fully justified, and they ought to be more rigorously enforced. It should be made clear that tuberculosis is not the only disease which is transmitted by expectoration, but many other diseases may be thus transmitted, so that nobody ought to spit on the floor of a house or public place. Furthermore, there are many tubercle bacillus "carriers" who do not suffer from the disease which they are liable to transmit, especially to infants and children. The fact that indiscriminate expectoration is prohibited irrespective of the question whether the offender is tuberculous or not, makes it easier to exact it from the phthisical patients, who do not like to be stigmatized.

In the case of children, especially infants, it is not only sputum which is dangerous, but also the droplets flying out of the mouth and nose during the acts of coughing, sneezing, and talking. For this reason a consumptive should not associate with infants, even if he is careful with his expectoration. Droplet infection may prove disastrous to infants. In the case of adults, coughing and sneezing are hardly dangerous. We have already mentioned Saugman's conclusion that it is not dangerous for adults to be coughed at by a tuberculous patient (see p. 149).

Cuspidors.—The disposal of the expectoration is therefore an important problem, and it has been suggested that the best means of render-

ing it harmless is that it should invariably be deposited in some form of cuspidor.

Floor cuspidors in rooms, especially in public places, are a nuisance; they cannot be tolerated in any decent home for both sanitary and esthetic reasons. They are unsightly, and just as much of the sputum is often deposited around the vessel as within it. Flies, cats, and dogs are frequent visitors, and with mouths or legs covered with sputum may proceed further in their quest for food, and deposit the bacilli on food which is subsequently used by the inhabitants of the house. The elevated cuspidors, of which we find such beautiful illustrations in a certain variety of books on tuberculosis, may be good for certain institutions, especially those harboring advanced consumptives, but they should not be, and are not, used in homes and public buildings. They are also an invitation to spit; they provoke expectoration in persons who otherwise would not do it. This is the reason why they are hardly seen anywhere, except in books and institutions.

The pocket sputum flasks are objectionable for other reasons. Their variety is great, if we are to judge by the large number illustrated in popular books on the prevention of tuberculosis. The ingenuity of the designers or inventors is noteworthy and could have been used to better advantage in other directions. They are, however, not used outside of institutions to any noticeable extent. I fancy that a person who would take out a sputum flask, even one of those which look like cigar boxes, lunch boxes, etc., and spit into it within the sight of people in a public place, would create a miniature panic among some who have read popular literature on the prevention of tuberculosis.

They are objectionable for another reason. No matter how wide-necked they are made, the patient must apply his lips to the mouth of the flask if he wants to deposit the sputum within it. The result is that part of the sputum sticks to the lips or mustache and beard, and this must be removed with a handkerchief. Even if all male patients would consent to shave clean it would not help. I have observed that the lips are very often covered with sputum after the patient has expectorated into any of these flasks.

In institutions they should be used, and the ones made of pasteboard, kept in a tin frame-holder, are the best. Patients in the advanced stages of the disease should use them at home in case they expectorate large quantities of sputum.

But I can see no reason for urging them on patients in the incipient stages of the disease, expectorating but little sputum. Physicians trying to imitate legislators who pass laws which they know cannot be enforced, defeat their own ends. We cannot induce a patient to carry a sputum flask with him, no matter how fine and deceptive its construction may be, and to use it in public. I have also known some patients in the incipient stages of the disease who left sanatoriums because they could not tolerate their fellow-sufferers walking around

with sputum cups in their hands. Advanced patients are hardened in this respect, as a rule.

Patients in the incipient or quiescent stages of the disease can empty their chests in the morning into cuspidors containing some cheap disinfectant. It should soon be emptied into the water-closet. Urging them to burn it is usually a vain effort, if only because there are no facilities in modern homes for the purpose. Those expectorating considerable quantities may efficiently dispose of their sputum by the use of paper napkins. Toilet paper will also answer the purpose. Several thicknesses are folded once, so as to receive the sputum; the paper is again folded and the ends folded over so as to enclose the expectorated material, and then placed in a grocer's bag (about 6 by 12 inches). The bag can be pinned to the side of the bed, or clamped to the small bed-table. Several times a day, depending on the amount of sputum, the bag and its contents should be burned, if there are facilities for the purpose. The folded paper pockets containing the sputum may, however, be disposed of by dropping them singly into the water-closet and flushing it immediately.

There is no question that there are valid objections to the handkerchief, though it is not so strong a menace as some writers would lead us to believe. But the average patient will use nothing else for reasons already stated. Portable sputum cups are used only in institutions and in homes, but, despite the agitation in their favor, we fail to meet persons in the streets or public places of any large city in the world, carrying and expectorating into them, although we know that thousands of consumptives are everywhere. Even if it is a compromise, we must submit to the inevitable and permit patients to use handkerchiefs. It is best that they should be made of gauze or cheap cotton, which may be destroyed after use; or they may be of Japan paper, which may be deposited into the water-closet which is immediately flushed. If made of better material, the handkerchief should be boiled before washing. Boiling is a better and surer bactericide, especially of tubercle bacilli in sputum, than any chemical disinfectant.

Duties of the Community in the Prevention of Phthisis.—In its demands on the consumptive to shape his life in such a manner as to prevent the dissemination of the disease, the community must not neglect its own duties to the unfortunate individual, who is suffering to a great extent because of conditions which the authorities have permitted to prevail. The community must not only provide shelter, proper nourishment and medical attendance for those patients who are not in a position to procure it at their own expense, but must also see to it that the conditions favoring the development of phthisis should be eliminated.

Laws regulating the sanitary and hygienic conditions of dwellings for the working people, among whom the proportion of phthisical patients is high, should be passed and rigorously enforced. Tenement house laws, passed and enforced, have a greater influence on the reduction of the morbidity and mortality from consumption than all the

lectures delivered in and out of season to social workers, policemen, teachers, and workmen, on the perils of the tubercle bacilli and the best means of killing them. The demolition of the old-style tenements with numerous rooms without windows has saved many more persons from developing phthisis than all the sanatoriums which are supposed to isolate the sources of infection, but which, in fact, exclude those in the advanced stages and permit them to come into intimate contact with infants and children. The abolition of the sweat-shops in New York City deserves more credit for the prevention of phthisis than all the leaflets which have been distributed by so many overlapping agencies, each eager to get at the persons who cough as a result of tuberculosis or some other disease and "follow them up."

Light and well-ventilated dwellings and workshops are of prime importance in preventing phthisis, and the community in which there are no rooms without windows and no sweat-shops or factories which are dark and badly ventilated has the least consumptives to care for.

Good wages and short hours, allowing good nourishment, and time for outdoor exercises and recreation, are important in the control of phthisis.

Marriage of the Tuberculous.—The problem of marriage is one which the physician often has to solve for his patients. We frequently have to answer the question whether a non-phthisical consort should continue to live with the phthisical partner; or whether a tuberculous patient, in any stage of the disease, may enter the married state. Answering these questions involves a consideration of several factors: The dangers of transmission of the disease to the non-phthisical consort; the dangers to the potential offspring; and the effect of the married state on the patient.

The dangers of transmission of the disease to the consort are negligible. We have brought statistics proving that the unaffected consorts of consumptives are no more liable to become phthisical than others of the same age and social condition (see p. 149). The unaffected consort has undoubtedly been infected during childhood, and reinfection is not likely. Whether he or she will develop phthisis depends on factors other than intimate association with a tuberculous consort. The conclusion is therefore justified that, as regards transmission of the disease alone, there is no more danger in marriage of phthisical patients than in cases of cancerous or diabetic patients. Our answer is to be about the same as when two persons who had both been previously infected with syphilis ask whether they are permitted to marry.[1]

[1] In this connection it is interesting to cite the following lines from Metchnikoff: "At the age of twenty-three," he says, "I married a young lady of the same age who was attacked by grave pulmonary tuberculosis. Her condition of feebleness was such that it was necessary to carry her in a chair in order to mount the few steps which led to the church where our marriage was to be celebrated. . . . My wife died of tuberculosis after four years of suffering. I passed the greater part of that time by her side in the greatest intimacy without taking any precaution against the contagion; nevertheless, in spite of these conditions, which were especially favorable for catching the disease, I have remained free from tuberculosis, and that during forty-four years since my marriage." (Bedrock, January, 1913.)

The danger to the children that may result from the union is enormous. If the newborn child will remain in the proximity of the phthisical parent, it will most likely become infected during infancy and succumb. Under the circumstances, unless they are satisfied to remove the child immediately after birth and not see it till it has passed the first two years of life, phthisical patients should not procreate. This is a point which cannot be emphasized too strongly to tuberculous patients who are married, or contemplate marriage. It is especially dangerous for an actively phthisical woman to raise infants. They will, we can say almost without exception, acquire the disease and succumb during the first year of life.

The effects of the married state on the patient are different in men, as compared with women. On the average male patient in the incipient or moderately advanced stages of the disease, sexual intercourse has the same effect as on the average person who is not in perfect health. If he indulges moderately, it does him no harm at all; in fact, it may be beneficial because it prevents brooding over enforced abstinence which is often seen among all classes of men. It also precludes venereal complications which may have an effect on the phthisical process.

With women, things are different. So long as they do not become pregnant there are no strong and valid reasons against married life. In fact, among the working classes the married consumptive woman is better situated than the single who soon after becoming tuberculous also becomes a dependent; and if she has no family to care for her, she is doomed. The dangers of pregnancy have been overestimated by many authors, as we have already shown (see p. 579). It is clear that women with active and progressive tuberculosis are in grave danger after becoming pregnant; this has also been observed to be true of experimental tuberculosis in animals. But the intensely chronic, quiescent, and arrested cases are hardly ever harmed by marriage and pregnancy. To be sure, it will be observed that a certain number, about 20 per cent, will suffer recrudescence of the disease during pregnancy and lactation, but taking a similar number of unmarried tuberculous women with quiescent or arrested lung lesions, we will have about as many reactivations during any two years which involve pregnancy and lactation. We have already given details as to the clinical criteria which may guide us while advising patients on these points (see p. 580).

Married women with active tuberculosis are to be given detailed instruction on the proper methods of prevention of conception. If they become pregnant the induction of abortion is indicated and justified both for the sake of the prospective child, which is bound to become tuberculous unless removed from the proximity of the mother immediately after birth, and for the sake of the mother, who is liable to succumb to acute or subacute tuberculosis soon after childbirth.

The demands made by some enthusiastic advocates of eugenics that tuberculous persons should be prohibited by law from marrying, has

no scientific basis in view of what has been stated above. The race is not in danger of deterioration because of children derived from tuberculous stock. We have already mentioned that tuberculous cattle have been used for breeding purposes by removing the calves immediately after birth. We see no reason why this should not hold in human beings. Moreover, prohibition of legal marriage does not exclude extramarital sexual intercourse and childbirth with their concomitants. Free instruction on the means of prevention of conception is more likely to eliminate phthisical stock, and thus prove of eugenic value, than prohibition of marriage.

On eugenic grounds it has also been stated that tuberculosis is rather a benefactor of humanity. It removes the weakly, the decrepit; in short, the unfit. In time, it is thus argued, all the susceptible will thus be removed and the race will improve. But we have seen that it is not only the weakly and decrepit which are likely to be attacked. The large number of athletic youths who develop tuberculosis in the prime of life prove that the strong suffer as often as the weak; the enormous number of intellectual giants who have succumbed to tuberculosis (see p. 297) show that humanity would be the gainer by eradicating this disease.

A patient presenting himself or herself with the problem of marriage should be explained the situation along the lines just detailed, and if he or she is intelligent, we may rest assured that the action will be reasonable for both the married people and the community. The ignorant and reckless will not consult us in such matters, and if they do, they will not follow instructions. For this reason, they should be left out of consideration in discussions of this kind. One thing I always insist on with my patients: The unaffected partner must be informed about the true state of affairs and given the choice. Very often it will be found that a good woman will greatly help along a consumptive toward recovery which could not have been attained if the patient had remained single; or that a female patient will recover after marriage to a man who gives her a good home, proper food, etc.

CHAPTER XXXV.

GENERAL MANAGEMENT OF THE CASE.

Should the Patient be Told that He is Tuberculous?—The diagnosis of pulmonary tuberculosis having been definitely made, there arises the question whether the patient should be told the true nature of his disease. Many physicians are inclined to keep him in ignorance as to the true state of affairs, and to tell him that he is merely affected with a "mild bronchial catarrh," "pleurisy," a "protracted cold," etc. Very often a patient is brought to the office by relatives and friends who beg the physician that in case tuberculosis is diagnosticated, the patient should under no circumstances be told the truth.

There are many valid reasons against such a procedure. From the standpoint of the physician's personal interest, it is bad practice. It is always to be borne in mind that the patient will, sooner or later, find out the truth and blame his doctor for deception, or more often, accuse him of ignorance and claim, with considerable justice, that had he been informed in time he might have taken better care of himself.

But there are reasons of more importance than the doctor's interest for telling the truth to every patient on such occasions. It must never be lost sight of that tuberculosis is transmissible, particularly to infants and children, and that the patient must be warned against the possibility of disseminating the seeds of the disease. This can only be done by telling the patient the true state of affairs, and giving him details of the principles of prevention. Moreover, the average patient knows that, in many cases, the chances of recovery diminish with the advance of the disease, and negligence in informing him of his opportunities at the earliest possible time may prove disastrous. We do not know of any quick cures, and the coöperation of the patient is absolutely essential. He can only take proper care of himself and those around him when he knows the true situation.

It is noteworthy that relatives and friends who have requested a physician to keep the patient in ignorance of the fact that he is tuberculous are always grateful in the end when he is tactfully informed of the truth.

Irrespective of requests of friends and relatives, the patient is to be told plainly and unequivocally that he suffers from tuberculosis. In really incipient cases this can be done in several instalments, because it usually requires several examinations to make a positive diagnosis. When finally told, it is to be emphasized that he is in the incipient and curable stage, and assurances given that in his case the prognosis

is very favorable. But it must be insisted upon that the patient's coöperation is absolutely essential to attain a cure. An intelligent patient may be given details of the nature of the disease and it may be pointed out that his own determination to follow instructions is of more importance than all the medicines and climates; in fact, without his own coöperation, he is lost even if he consults the best known specialists, enters the most famous sanatorium, or emigrates to any climatic resort. It is a striking fact that nervous and excitable patients who are expected by their relatives to break down on hearing the truth, resign themselves to their fate and often display courage and determination worthy of heroes.

"Unless we carry conviction to our patients," says Arthur Latham,[1] "they are unlikely to put up with the restrictions which are inevitable to proper treatment. It is a disastrous thing to talk about a "weak spot" in the lung. It is our duty, in an overwhelming proportion of cases, to state his position frankly to the patient, to explain intelligibly the reasons for the treatment prescribed, and the possible penalties which may have to be faced if our advice is neglected. If we can convince our patient, we shall in all probability have won his loyal coöperation, which is half the battle; if we fail to convince him or get him to see the reasonableness of our advice, we cannot expect to find treatment carried out with sufficient earnestness and consistence to be of real value."

The suggestion has been made by Penzoldt[2] that the dose of truth given to the patient should be in inverse ratio to the seriousness of the case—the less the chances of recovery the smaller the dose of truth. In incipient and hopeful cases the whole truth is best, but the term "consumption" should be avoided in all cases; "tuberculosis" is a term which covers everything for the patient, though as we have seen, it is not exactly correct scientifically or clinically. But in the popular mind it has been of late considered a hopeful and curable disease, if taken in time. Some patients may be told that when neglected, "tuberculosis" may turn into consumption.

It is different with advanced and hopeless cases. They present themselves asking whether their cough is really due to consumption and it is at times a pity to tell the unfortunate patients the true state of affairs; not unless we are not averse to shortening their days. Still, for obvious reasons it is always imperative that some relative or friend should be told the truth. Similarly, in cases of acute or subacute pulmonary tuberculosis, or in progressive cases with complications, such as those suffering from diabetes, tuberculosis of the kidneys, etc., in addition to the active pulmonary lesion, it is often advisable to console the unfortunate and doomed patient if he likes it, by telling him that the prognosis is excellent.

[1] Practitioner, 1913, **90**, 38.
[2] Handbuch der Therapie, 1910, **3**, 205.

Economic and Social Conditions.—In outlining the treatment to be pursued, the social and economic condition of the patient are always to be borne in mind. *It is not advisable to tell a patient of limited means that a certain private sanatorium, or a climatic resort in a distant part of the country, is good for him.* He is likely to brood over the fact that owing to his poverty he is lost, when in fact he could get along very well at home or in the neighborhood of his city. Well-to-do patients may be sent out of town with only suspicious symptoms and signs of the disease on the principle of some physicians to treat all "suspects" as tuberculous until proved to be free of the disease. The rest during the vacation does them good; in fact, they usually need it. But patients with limited means should never be treated in this manner. In them only a positive diagnosis of tuberculosis should be the criterion for radical and costly treatment.

Relation of Physician to Patient.—A great deal has been written about the relation of the physician and his tuberculous patient, and it has been repeatedly stated that the former must possess certain qualifications which, if taken seriously, would exclude 99 per cent of practitioners from the category of physicians competent to handle an ordinary case. According to one writer, the physician must possess no less than an extraordinarily strong personality, immense will-power to impress it on his patients, unusual teaching ability, fervent enthusiasm and unremitting interest, etc., if he is to meet with success.

Evidently these requirements are such as all ideal physicians should possess if they are to be fit for successful practice. The truth is that in most cases it is quite easy to gain the confidence and coöperation of the patient, if this is at all obtainable. The main problem is to retain it for the long period of time it takes until the termination of the case. This is especially true of chronic phthisis which runs an undulating course with accidents (hemorrhages, fever, anorexia, etc.) which come and go unexpectedly, and are liable to shatter the most implicit confidence. This is one of the reasons why tuberculous patients, next to those suffering from venereal diseases, are the best prey for quacks and charlatans.

My observations lead me to the conviction that the average tuberculous patient can be easily managed and his confidence retained for an indefinite time when we appeal to his reason. It is a grave mistake of many superintendents of public sanatoriums who try to obtain the coöperation of their patients by keeping them in constant fear of punishment—expulsion. As one patient told me, the superintendent inflicted severe punishment on patients for small infractions of the rules of the institution because for these dependent patients the only hope of recovery was the sanatorium. Such severity does not at all help along in gaining the confidence of patients. I know of public sanatoriums in which the patients are always coerced into obedience of the rules and to submitting to prescribed treatment, but they do not discharge the proper proportion of cured patients, and a very

large number leave the institutions of their own volition before the physicians discharge them.

To a certain extent the patient treated by his physician at home is more amenable to reason than those in public sanatoriums. The physician in private practice is in a position to individualize his cases and more easily persuade them that their only chances for recovery lie in their implicit obedience to orders. When the patient is told the reason why we want him to rest the greater part of the day for weeks or months; why we want him to eat certain kinds and quantities of food; why we want him to submit to the operation for artificial pneumothorax, etc., he is more likely to submit than when we threaten him. All this can be done with alleged ignorant patients, who usually have more common sense than they are credited with, as well as with the intelligent and cultured. In fact, the former are, as a rule, more tractable than the latter. We must always remember that these patients make great sacrifices for months, and need consolation and encouragement which only the reasonable physician is able to bestow.

Personal Hygiene.—The first instructions given to the patient are as regards his personal hygiene. This can best be done only after careful inquiry into his daily habits which, as a rule, are found not to have been exemplary; otherwise he would not have been likely to develop phthisis. To be successful, it is necessary to enter into the smallest details of every-day life, and most patients appreciate it greatly.

Treating patients in cities, after deciding against a sanatorium, it is of immense importance to ascertain their home surroundings. A call should be made at the house of the patient to see whether it is fit for a tuberculous individual, and especial attention should be paid to the location of the sleeping room, its size, windows, exposure, etc. In case these are not found satisfactory, moving should be urged, preferably to the outskirts of the city or a suburb. Details are given in Chapter XXXVII.

In our attempts at adapting the patient's mode of life to the therapeutic indications, we meet with great obstacles when trying to impress him with the urgency of cessation of work, physical and mental, and it is particularly difficult to persuade patients with mild lesions showing few constitutional symptoms. They are convinced that work does them no harm. The poor point to the necessity for providing for themselves and those dependent on them, while the well-to-do are apt to be even more intractable in this regard. They must not neglect their business, they must finish some task they have undertaken, they are deeply absorbed in some studies; they must continue at college until graduation, etc. But the careful physician is not moved by these pleas and points out to the patient that just because he is in such good physical condition the prognosis is so good. But should he continue working physically or mentally, the disease will surely make inroads on his vitality and the chances of ultimate and

complete recovery will vanish. Whether he leaves the city or not, the patient may be induced to take a complete vacation with all the separation from the activities of life a vacation entails, but without any of its pleasures. The details about rest and exercise are given in Chapter XXXVI.

Baths.—The mortal fear for "colds" entertained by many is accentuated as soon as the diagnosis of tuberculosis is made and one of the first results is that the patient ceases to bathe. In many advanced cases, or even in incipients who suffer from profuse nightsweats, large patches of pityriasis versicolor are to be seen on the skin of the neck and trunk. When told that bathing will remove it, women are easily induced to take frequent baths. But all are to be instructed that bathing improves the circulation, activates the skin, and invigorates the individual. It must be insisted upon that the patient bathes frequently and follows it up by vigorous rubbing of the skin with a rough towel.

The question of cold baths in tuberculosis has been very much debated. In some institutions, cold baths and frictions are the chief elements of the cure. They are urged for the purpose of hardening the body against colds. But many are not fit for the purpose of hardening; they do not react properly and, instead of feeling refreshed and invigorated after a cold bath, their extremities are livid, benumbed, chilled, and they feel altogether miserable. These patients, independent of their physical condition, are better off when taking only warm baths, twice or thrice weekly, followed by frictions. The statements of some that every tuberculous patient can be subjected to a process of hardening, if methodically applied, does not hold, as is evident from the fact that it is not pursued systematically in most sanatoriums. Bed-ridden patients may be sponged with tepid, or even cold, water during febrile attacks with great benefit. Patients who have been in the habit of taking cold baths, douches, or sponging, every morning should continue to do so during their illness, but those who do not bear these procedures well should only bathe in warm water, as was just stated.

Robust patients may also be allowed swimming within reasonable limits; bathing outdoors, especially sea bathing, is good for quiescent cases. Turkish and Russian baths are decidedly harmful in active cases.

Clothing.—The tuberculous patient should be sensibly clothed, the aim being to keep him warm during the cold winter, but not overheated. The fear for "colds" is responsible for the excessive underwear which we often find on patients, and, coupled with the several vests, sweaters, coats and overcoats, they are often fairly borne down by the weight of their clothing. The well-known red flannel pad, "the chest protector," has not as yet been abandoned after all the medical agitation against it; we often see patients wear them, and every drug store sells them. Not only do the poor and ostensibly ignorant classes

make use of them but we meet them among so-called educated patients. They become habituated to this excessive covering of the chest, and perspire freely. When they attempt to remove it they are easily chilled, which is responsible for many of the catarrhal complications which occur during the course of the disease.

In the beginning of the treatment, the patient is to be discouraged from such practices. He is to be told with due emphasis that woolen underwear, of thickness consistent with the season of the year and other meteorological conditions, is all that is necessary. A woolen garment has a capacity for absorbing considerable moisture without feeling wet, while cotton soon becomes saturated with moisture. If evaporation takes place suddenly, the body is chilled. Some patients are unduly irritated by wool next to the skin, but by constant wear they overcome this difficulty. Of course, it is important that the underwear worn during the day should not be worn during the night.

All sudden changes in temperature within and out of the house are to be met by changing the overgarments. During the winter a fur coat is good, and can be purchased for about the same price as a good overcoat. Those taking outdoor treatment on a reclining chair need extra wraps during the winter. Carrington[1] gives a complete description of the various appliances which may be used for the purpose.

Women are less easily managed in regard to clothing than men. The low cut around the neck and chest is very harmful to tuberculous women, and they are to be induced to forego some of the fashions in vogue. But what is of most importance is the corset which many refuse to part with, claiming that it is not at all the figure they care for, but that they have been habituated to stays and feel uncomfortable without them. But when explained in detail the way a corset, even of those called "hygienic," interferes with the respiratory movements of the thorax, most women submit to the argument.

Smoking.—The problem whether a patient who has been found tuberculous should give up smoking has troubled many physicians in sanatoriums. Some have been inclined to prohibit it indiscriminately and failed, as a rule. One who has been habituated for long years to smoking cannot easily give it up, and when he does he is often so nervous and miserable that it has an immense influence on his general well-being and the course of the disease. The fact is that *smoking has no deleterious influence on the tuberculous process in the lungs*, and there is no reason for imposing an additional hardship on the patient. Of course, chewing tobacco should be prohibited.

The assumption that smoking predisposes to tuberculosis and aggravates the pulmonary condition if indulged in by tuberculous individuals, has been shown to be incorrect. Gerald B. Webb,[2] in a statistical investigation, found that of a comparatively large number of soldiers in the United States Army, the proportion discharged from

[1] Journal of Outdoor Life, 1912, **9**, 262.
[2] Am. Rev. Tuberc., 1918, **2**, 25.

active service because of active pulmonary tuberculosis was no higher among those who smoked than among those who did not. His conclusion that inhalation of the smoke of cigars or cigarettes does not predispose the lungs to tuberculous disease confirms this fact which has been long ago observed by clinicians. Webb, however, found that but few non-smokers have rhonchi, or sibilant rales, while the majority of smokers do present these signs of bronchial irritation. But as has been pointed out by Krause[1] in this connection, inflammatory processes have not been found to be predisposing factors for bacterial infection; they may rather be considered factors in the resistance against infection. William S. Duboff[2] found that tobacco does not predispose to laryngeal tuberculosis, and that throat complications are no more frequent in tobacco users than in those who use no tobacco. Laryngitis, of specific character or not, appears to be equally as common among women as among men in the course of pulmonary tuberculosis, showing that tobacco is not an important factor.

When there are laryngeal complications smoking is apt to cause irritation and cough. However, I am inclined to follow Fetterolf's[3] suggestion: The patient, if he craves for his cigar, cigarette, or pipe, is thus instructed: "The smoke is not to be blown through the nose or inhaled; that if a cigar or cigarette is used it shall be smoked in a holder at least four inches long, and, finally, that the smoking be done in the open air. The main evils, barring excess, are dry heat and dust which are drawn into the pharynx and larynx. This is of greater significance the shorter the smoked article grows, and if the cigar or cigarette is used in a holder and only the first half is smoked, this evil is largely done away with." It is Fetterolf's belief that with such precautions as just mentioned and with the smoking done in the open air, no harm will result. A non-smoking patient in a close room with others smoking is at a greater disadvantage than one who is smoking in the fresh air.

Occupation.—A great deal has been said of occupations fit for tuberculous patients. The problem is not one which concerns those with active disease, but the convalescents, as well as those who have recovered. A patient during the active course of phthisis in any stage should have no occupation at all. He cannot work, he must not attend to any vocation which requires physical or mental exertion. Mistakes are often made in permitting patients in the incipient stages to wind up their business, to finish a course in a school, etc. This is a point which will be discussed later on while speaking on rest and exercise and cannot be emphasized too strongly.

It is very difficult to advise patients who have recovered from phthisis as to their future activities in the affairs of life. With the rich and prosperous the matter is very simple: They may be allowed to return

[1] Am Rev. Tuberc., 1918, **2**, 99.
[2] Ibid., **2**, 21.
[3] Hare's Modern Treatment, **2**, 405.

to their vocations provided they know how to take care of themselves. Under supervision, and with careful observation of the ordinary rules of healthy life, they very often avoid relapses. The same is true of professional people who can resume their life work, perhaps at a slower pace. But with those who have been artisans, manual laborers, etc., especially in "precarious occupations," the matter is different. It is, indeed, easy to advise one to change his vocation, as is done in sanatoriums when patients are discharged, but whether the patient is more harmed by working at his trade and earning for his support, than by starvation because of lack of funds to buy food, pay for his lodging, etc., is hard to decide.

Moreover, a change of occupation is not feasible in the vast majority of cases, especially with skilled artisans. They cannot easily accept low wages when at their own trade the pay is much higher, and the hours shorter. It is also a fact, only rarely considered by medical men, that the artisan has usually adapted his organism to his peculiar occupation; in fact, there is a process of selection going on, certain persons are attracted to certain trades at which they succeed. They must return to these occupations after recovering from the disease, if they are at all to be able to support themselves. And they do, in fact, in spite of our protestations.

But we must try to keep convalescing tuberculous patients from hard muscular exertion, if relapses are to be avoided. They are to be under medical supervision for several months after beginning to work, and if they show any signs of damage to their constitution, especially fever, dyspnea, tachycardia, etc., they must stop before it is too late. Nor should a cured patient be allowed to work at any dusty trade, such as pottery and earthenware manufacture, cutlery and file making, certain departments of glass making, copper, iron, lead and steel manufacture, stone cutting, textile trades, fur- or cigar-making, iron-grinding, etc. We have seen the effects of organic, mineral, and metallic dust in the direction of engendering a soil susceptible to phthisis. When we bear in mind that a patient with cured tuberculosis almost always harbors virulent tubercle bacilli in the cicatrized area of the lung, we can easily understand that irritating dust may at any time flare up a dormant lesion into renewed activity or cause metastasis.

It is always to be remembered that farming is not the only outdoor work, nor is it the best. Farm laborers usually work very hard for long hours, small pay, and with food that does not satisfy the majority of city dwellers. In addition, as has been pointed out by Vogeler,[1] the lack of amusement during the hours of recreation and the enervating heat during the summer are serious drawbacks. Of course, it is different when the patient can raise funds to buy or lease a farm for himself.

There are in cities many more or less remunerative occupations

[1] Trans. Nat. Assn. Study and Prev. Tuberc., 1912, **8**, 113.

which are suitable for this class of cases, as conductors, motormen, ticket agents, attendants at ferries, watchmen, solicitors, etc. My observations lead me to the conviction that workers at the garment industries, excepting at fur, may safely return to their occupations, provided they find employment in light and well-ventilated workshops. The same is true of the building industry, provided the exposure to the vicissitudes of the weather is not excessive nor the hours too long; and of clerks, salespersons, etc. Indeed, I have been struck with the fact that *when a patient who recovered from phthisis is unable to pursue the vocation for which he has been trained for many years, he will not do well, even if he remains idle indefinitely.*

In advising tuberculous convalescents about occupations, the fact must not be lost sight of that we know very little about the subject. We have shown that only certain kinds of dust predispose to phthisis, while others, on the contrary, apparently confer more or less immunity against the disease. Among the latter may be mentioned coal dust, lime dust, etc. (see p. 132). The same is true about the problem of indoor as compared with outdoor occupations. When we find that hotel servants have a very high mortality from tuberculosis, it does not necessarily mean that it is because of their indoor work. On the one hand the work, not involving strong muscular exertion, attracts weaklings, and then also they are liable to drink excessively. Street sweepers are apparently spared by tuberculosis to some extent, but their outdoor occupation also involves the inhalation of dust excessively. I have seen very few tuberculous patients among the workers in the underground subway of New York City despite the fact that they are employed in an indoor occupation *par excellence*. The same has been noted in London. Cobbett[1] mentions that the old underground railway in London, before it was electrified, was considered as a particularly favorable place for consumptives. The stations of Portland Road and Gower Street, which were entirely below the surface of the ground, and which were formerly notorious for their mephitic vapors, were regarded as the best for tuberculous patients, and the authorities transferred to these stations any of their workers who showed signs of incipient tuberculosis. The problem of "indoor," as compared with "outdoor" occupations is thus seen not to be as simple as some believe it is as regards tuberculosis.

In judging a patient with a view of selecting an occupation for him, we may be guided by the condition of his temperature, pulse, respiration, and general constitution, but the extent of the lesion is a hazardous criterion. All who have had experience agree with H. M. King that "it frequently happens that a satisfactory condition of health as determined by restoration of working efficiency maintained for many years is not incompatible with physical signs which of themselves would indicate active disease." I have seen many cases in which the

[1] Causes of Tuberculosis, London, 1917, p. 98.

reverse was true, the patient showed no signs of active disease in the lung, yet as soon as he began to work he broke down with fever, rapid pulse, dyspnea, etc. These patients cannot work at all. Then there are others who will work for several months and, owing to an evanescent, acute, or subacute exacerbation, are laid up for several days or weeks. With these it is very difficult to judge the ability to work. All tuberculous patients, even after completely recovering from the disease, find it difficult to compete with healthy persons, but the class just mentioned is more apt to lose in the struggle for existence. They must find for themselves employment of a nature which makes them independent of strict regularity.

On the whole, it appears that cured patients do best when returning to their old vocations for which they have been trained, and at which they can earn the most with the least possible effort. It may be said that, with some striking exceptions, if a patient is not able to pursue his former line of work he is altogether disabled.

CHAPTER XXXVI.

THE REST CURE.

Principles of the Rest Cure.—Nature makes a strong effort at repairing the affected lung in tuberculosis, but we only rarely appreciate the method it pursues while doing it. Examining the chest of a tuberculous patient, we find on inspection that there is a strong tendency to putting the affected area of the lung at rest. As already has been shown, during the early stage the muscles overlying the pulmonary lesion are almost invariably rigidly and spasmodically contracted. This contraction has been ascribed by Rubel[1] to the physiological coördination of the respiratory center. It inhibits or prevents the motion of the underlying diseased lung to a certain extent. Later, pleural adhesions are formed which impede the respiratory movements of the lung to a yet greater extent, as is seen in the lagging of the affected side of the chest, offering favorable conditions for cicatrization. This immobilization of the affected part of the lung also slows the circulation of blood and lymph in that area, retains the bacteria and their toxic products, thus lessening toxemia and preventing metastatic auto-infection of unaffected parts of the lung. Rubel has shown experimentally that functional rest greatly contributes toward a cure of tuberculous lesions in the lung. He immobilized one lung in rabbits and then infected them by the intravenous way. In the relatively immobilized lung the lesion was found to be of the chronic and favorable variety, while in the freely movable lung it was acute and progressive.

Surgeons have utilized physiological and functional rest in the treatment of tuberculosis of bones and joints. The modern treatment of Pott's disease, and tuberculosis of joints in general, consists mainly in affording rest to the affected parts. The splint has done better than the knife in these forms of tuberculosis. Formerly physicians aimed at procuring rest in tuberculous diseases of the thoracic viscera by the application of strips of adhesive plaster, thus immobilizing the thorax; and at present the induction of an artificial pneumothorax puts the affected lung at complete functional rest. "In breathing, a normal person 'opens and shuts' the lungs nearly 30,000 times a day," says Webb. "By rest we aim to make the breathing as shallow as possible, imitating almost that of hibernating."

In febrile cases rest has a rationale which is clear to everyone who gives some thought to the subject. Fever is an indication of activity

[1] Ztschr. f. Tuberk., 1908, **10**, 193, 319; Roussky Vratch, 1907, **6**, 648, 721, 750, 896.

of the tuberculous process and results from absorption of toxins and decayed lung tissue. By keeping the patient at rest we reduce the frequency and depth of respiration, and thus less of the toxins are washed into the blood stream and the fever declines. Fever also means increased metabolism and we must not further increase it by exercise. With the reduction in the fever there is an amelioration in the cough, and an improvement in the appetite, resulting in better nutrition of the patient.

Rest and Exercise in Phthisis.—In former days the treatment of tuberculosis consisted mainly in removing the patient to some country place, or better yet, to an institution, and urging him to exercise in the open air. Thus, the main principles of the treatment in Brehmer's sanatorium were outdoor exercise for long hours, daily walking, driving, horseback riding, mountain climbing and respiratory exercises. The same methods were followed in institutional and home treatment by many physicians until about thirty years ago.

The development of sanatoriums in which careful observations have been made on the effects of these exercises on tuberculous patients has resulted in swinging the pendulum, and rest has come to the foreground as the most important factor in combating the disease, so that at present vigorous protests are heard from many sides that the indolent life led by sanatorium patients is often more harmful, for various reasons, than the exercises which were formerly in vogue. Indeed, Paterson reports just as many cures at Frimley, where the patients do graduated work, as in sanatoriums in which they are kept at perfect rest for long months, or even years. Moreover, a curious phenomenon is to be observed in most sanatoriums: The physicians, nurses, etc., are mostly recruited from the ranks of the tuberculous. They all work, more or less. Yet, on the whole, they are doing better than the patients who take the rigid rest cure. Of course, they are a select group, with chronic lesions. But patients with such lesions and activity are often kept at the rest cure for exceedingly long periods of time.

The contradictory evidence in favor of rest or work is obviously due to the fact that neither rest nor exercise is a panacea which will help in every case, but that each has its indications and contraindications. When patients presenting symptoms of active and progressive phthisis—fever, anorexia, emaciation, etc.—are urged to work or exercise, considerable harm is often done, and a favorable case may thus be converted into one which is decidedly hopeless. In the later stages of the disease, when the lesion has localized itself and the patient has no fever, eats well and feels strong enough to do some work, perfect rest may be distinctly harmful, as will be pointed out later on. Rest and exercise have their indications and contraindications.

Indications for Rest.—Nature puts most patients who suffer from active and acute forms of the disease at rest. They are weak, anemic, emaciated, and the exhausting cough, the dyspnea, and the phenom-

ena of toxemia in general, preclude any kind of exercise. But in the chronic cases, or even in some of the subacute cases, the patient may not realize his plight and continue working at his occupation until he breaks down, when it is too late to recoup the lost flesh and forces. Rest, properly applied, in this class of cases may prove life saving.

It is clear that *all active cases with fever, tachycardia, anorexia, emaciation, weakness, etc., are to be kept strictly at rest until most of these symptoms have disappeared.* It must be stated at the outset that the extent of the lesion is no reliable criterion as to the indications for rest and exercise. A patient in the incipient stage, with a limited and circumscribed lesion at one apex, and suffering from fever, dyspnea, anorexia, etc., is often more harmed by work or exercises than one in the advanced stages, with extensive involvement of both lungs, but with normal pulse and temperature.

With but few exceptions, *the rate of the pulse is as good an index of the fitness of the patient to work as there is.* So long as it is 90 or over per minute, or it is accelerated to that rate by mild exercises, the prognosis is not good, unless the patient is kept at perfect rest. In tuberculosis we often meet with unstable tachycardia; the pulse runs up to 120, or more, per minute at the least exertion or excitement. Such patients are to be kept in bed, or on the reclining chair, until we find that mild exercise, like walking slowly on level ground for a half or one mile, does not unduly accelerate the pulse. Some of these cases with tachycardia are afebrile, the temperature is in fact very often below normal, and exercise may not affect it, but the pulse is accelerated on the least exertion.

Dyspnea, when present, is another sign that the patient must be kept at rest. We must be guarded and not wait for subjective dyspnea, because many tuberculous patients have adapted themselves so well to their difficulties in breathing that they are not much disturbed by it, and when seen to breathe very superficially and rapidly, even more than thirty times per minute, they may inform us that they suffer no inconvenience in this respect. It is objective dyspnea which should guide us in our estimation of the effects of rest or exercise in tuberculous patients. Likewise, acrocyanosis is not to be taken as a sole criterion. Many tuberculous patients with healed lesions remain with bluish finger nails due to vasomotor disturbances. In some of these patients the circulation, while below par, is fair enough for ordinary activities.

Fever has been considered an indication for rest by most writers on the subject; in fact, *the problems of exercise and rest have usually been solved by the thermometer.* In cases of tuberculosis in which the temperature reaches 100° F. the patient is put to bed, and kept there until it descends to normal. In acute cases, with continuous fever, or during acute exacerbations in chronic cases, or when some complication ensues, such as pleurisy, or any non-tuberculous infection, complete rest is enjoined until the fever abates. In far-advanced cases

with hectic fever, reaching a high degree in the afternoon or evening, and dropping to normal, or even below, in the early morning hours, the patient is to be kept in bed at absolute rest. There are, however, cases of tuberculosis with fever which do not require strict rest. They are discussed in detail elsewhere, while speaking of the treatment of fever. It may be stated here that the body of many tuberculous patients adapts itself to the temperature, and moderate rises may not at all disturb them. In fact many of this class of patients do better work when the temperature is one or one-and-a-half degrees F. above normal.

Technic.—The rest cure, when indicated, is to be carried out methodically. In acute progressive cases it means complete rest in bed until the temperature declines to below 100° F. Some patients revolt, saying that they feel strong enough to walk around for several hours of the day, that they are lonesome and would surely improve if they were permitted to assume the erect position for some time. But they are to be told that fever cannot be cured outside of the bed, and as Poujade said: "Undoubtedly prolonged rest in bed weakens a patient, but it weakens less than fever, which kills."

In the home of the patient it is advisable, when feasible, to have two beds, in one of which he sleeps during the night, and in the other he spends the day. Considering that the patient may have to remain in bed for weeks or months, the enforced solitude is hard on him, and the change of the bed has some salutary effect. Moreover, these patients are apt to sleep during the day and suffer from insomnia during the night. One of the reasons for sleeping during the day is the extreme to which the rest cure is often carried. The patient is prohibited from reading, or accepting visitors. There is no reason for these prohibitions in those with mild fever, especially in tuberculosis which does not strike the patient down to the same extent as fevers due to other causes. By changing the room and bed they often become habituated to sleep in one bed, and remain awake during the day in the other. One room and bed may also be aired while the other is used.

In the morning, when the patient wakes, he is to be given a sponge bath—one with alcohol is invigorating—and dressed, the lower half of the window opened and the bed placed in such a position that he can look out on the living world. If he feels cold, a hot-water bag should be placed at his feet. Great care must be taken to prevent bedsores in prolonged and advanced cases.

When the temperature descends below 100° F.; in prolonged cases when it reaches this degree only at a certain time in the afternoon, but is near normal during the rest of the day, the patient may be kept at rest on a reclining chair during the greater part of the day, preferably outdoors, and reading and mild games may be allowed; only during the hours when the rise in temperature is expected is he to be made to go to bed. When we find that this does not increase

the fever, he may be permitted mild exercises, such as short walks, and the effects should be watched. We are often surprised to find that the fever disappears altogether with mild exercises. I have observed this to be the case when walking promotes expectoration.

This rest in bed is at times very difficult to carry out. The poor are often working for weeks while the temperature is high—I have seen them working with fever of 103° F., and even higher. When beyond control in this regard, the patient is to be sent to an institution, or to one of the day and night camps. I have seen excellent results in such cases after the patient has been at one of these institutions for a few months. Not only has the fever disappeared, but the patient was educated to appreciate the dangers of exercises during the febrile stage. But the well-to-do are not better in this respect. Very often we find them walking around, and even dissipating, in spite of the fact that their temperature is above 102° F. Indeed, they are often less amenable to reason in this respect than the poor. They are to be impressed that all business and pleasures are to be given up when the temperature is high.

Contraindications.—It was one of the great mistakes of many sanatoriums to urge all patients to keep at perfect rest and abstain from work or exercises, irrespective of the form of the disease and the constitutional symptoms. The result was that they turned out lazy people—hypochondriacs—who feared work, and who at the least fatigue considered themselves harmed by it after they had been cured. In most sanatoriums of today strong efforts are being made to avoid such mistakes.

As was already stated, the extent of the lesion is not always an index as to the indication for rest. There are many patients with extensive lesions in the lung, in fact with large excavations, who are well able to make themselves useful along certain lines. Indeed, there are cases in which prolonged rest is distinctly harmful. The nervous system may be functionally damaged beyond repair, the desire for activity may be stifled, and the resistance of the body in general may be lowered. It has also been suggested by Paterson and Inman that prolonged rest deprives the patient of certain reactions which bodily activity calls forth in the pulmonary lesions, and which are of great use in combating the deleterious effects of the disease.

In some sanatoriums where the rest cure has been carried to excess we often meet with patients who, after remaining in bed or on the reclining chair for several months, become mentally tired and listless; they lack interest in current affairs; other become hypochondriacs, consulting the thermometer several times a day and are alarmed at each finding above or below normal. They often lose all hope of ever getting cured and this despondency contributes greatly to the unfavorable course of the disease. Having met many patients with the "thermometer habit," I have often regretted the invention of this instrument of precision.

The graduates of sanatoriums in which the rest cure is carried to excess are apt to be lazy for the rest of their lives. Some of them, discharged from one institution, immediately seek admission to another. As Herman M. Biggs says, "A sick workman is converted into a healthy loafer." They fear muscular exercise of any kind and imagine that the least work aggravates their condition. In the State and municipal institutions in this country we find many with a record of having been in several sanatoriums. In fact, prolonged rest disables any human being, because the joints become stiff and any attempt to walk produces muscular weakness, pains and aches in the limbs. In some, the long rest favors the deposition of fat, which is very encouraging, but when carried to excess, which is not a very rare phenomenon among the tuberculous, it may disable the patient as much as active phthisis. These patients must have exercises to reduce the fat. This is mainly seen in patients in whom the disease may or may nor be active, but at any rate is not progressive; the lesion has become quiescent, completely surrounded by connective tissue. Rest may only produce obesity of various degrees, but does not assist in the healing of the disease focus in the lung. It is in these cases that graduated work or any exercise will do more than rest, and McLean's aphorism, "If the phthisical patient would live, he must work for it," is confirmed.

Exercise.—When the temperature and pulse become normal and remain so for several days, walking exercises are to be commenced, with a view of preventing the deleterious effects of idleness, as well as provoking mild reactions—auto-inoculations, which are, in most cases, of immense benefit. At first the patient is allowed to walk a mile on level ground and the effects on the temperature and pulse are watched. It may be done during the morning hours, when the temperature is normal, while in the afternoon, when there is some fever, the patient is ordered to rest on a reclining chair, or even in bed. But in those in whom the afternoon temperature is mild, below 99° F., even this precaution need not be taken, provided the pulse is below 85 per minute.

The following schedule for walking exercises, modified after that given by E. Hyslop Thomson,[1] may guide the patient who takes his own temperature:

Morning temperature at 7 A.M.	98.5 or lower; long or medium walk. 99.0; short walk. 99.5; rest outdoors or short walk around house. 100.0 or higher; remain in bed.
Temperature at noon	99.0 or lower; medium or short walk. 99.5; short walk. 100.0 or higher; rest in bed or reclining chair.
Evening temperature at 7 P.M.	99.5; only short walk on the following day. 100.0 and above; complete rest during following day.

[1] Consumption in General Practice, London, 1912, p. 223.

Hill climbing, or walking long distances, up to fifteen miles a day in afebrile cases without tachycardia may be permitted. The author has thus tested patients as to their ability to work, and was surprised to find often that they were rather invigorated by the exercise and they were then allowed to work for their support. Our patients are told to come to the office on foot, walking a mile or two, and if when they arrive the pulse and temperature are found normal, they are told to walk a longer distance the next day, etc. When this test shows that no harm is done by the exercises the patients are allowed to work, first under supervision, and later completely discharged with instructions as to the signs of danger.

Graduated Labor.—Practitioners among people in large cities are often impressed with the capacity for work of many consumptives amid unfavorable surroundings for years without visible harm. Among these cases there are many who are evidently active but not progressive: some are entirely quiescent. We must repeat that the extent of the lesion is less of an index as to the capacity for work than its activity as revealed by the constitutional symptoms, such as fever, tachycardia, dyspnea, etc. Paterson[1] developed his system of graduated labor after observing such cases in England. "It occurred to me," he says, "that if some consumptive persons under adverse circumstances and without any medical guidance could act thus without apparent injury, they ought, under ideal conditions and with the work carefully graduated in accordance with their physical state, to be able to undertake useful labor. On this assumption manual work should be of great advantage to patients undergoing treatment in a sanatorium, as at first it would do much to meet the objection that members of the working classes are liable to have their energy sapped, and to acquire lazy habits by such treatment; second, it would make them more resistant to the disease by improving their physical condition; and third, it would enable them by its effects upon their muscles to return to their work immediately after their discharge."

With a view to developing the muscles of the upper limbs, which are supposed to have more direct influence on the expansion of the lungs, Paterson[2] is not satisfied with walking alone. When a patient is found to be able to walk two miles a day without aggravating his condition, he is given a basket in which to carry mould for spreading on lawns, etc. No case of hemoptysis or of pyrexia occurred among these patients. When they have been on this grade with nothing but beneficial results for from three weeks to a month, they are given boys' spades with which to dig for five minutes, followed by an interval of five minutes for a rest. After a few weeks, several of the patients on this work, who were doing well, were allowed to work as hard as possible with their small spades without any intervals of rest. As they had all improved on this labor, larger shovels were obtained,

[1] Sixth Intern. Cong. Tuberc., 1908, 1, 886.
[2] Ibid,, 901.

and it was found that these patients were able to use them without the occurrence of hemoptysis or of a rise in temperature. About this time many of the patients were feeling so well that it became necessary to restrain them from doing too much.

Paterson worked out a schedule for graded work which brought excellent results. It was noted that many patients on their arrival are somewhat remarkable for a somewhat sullen and apathetic attitude, but as soon as their physical condition undergoes amelioration, all traces of gloom and depression leave them and they become lively, cheerful individuals. In many cases in which the improvement was not prompt, the effect of harder work was tried and often a progressive improvement was noted at once. Paterson found that the danger signals are: a temperature of 99° F. or higher in men and 99.6° F. in women, loss of appetite and slight headache. As soon as these appear the patient is to be put to bed until the temperature goes down to normal. In my experience, a rapid pulse is of even more importance as an indication that exercises are deleterious.

Inman, working with Wright's method of ascertaining the opsonic index in patients under this graded work system of Paterson, found that it was at some part of the day well above normal and he explains it as due to the stimulus supplied by the work, inducing artificial auto-inoculation; that the organism responds by the production of immune bodies. In fact, whenever excessive auto-inoculation takes place harm is done. This, he points out, must be readily recognized clinically if harm is to be avoided. "A patient doing well on the grade of work prescribed for him and with no abnormality of temperature suddenly complains of feeling tired, of loss of appetite and of headache; and the temperature chart registers an elevation to 99° or 100° F. These are precisely the symptoms which are found during the negative phase after excessive dose of bacterial vaccine."

Paterson is guided in his conduct of a case by the thermometer, and whenever the temperature registers 99° and over in men and 99.6° in women (by mouth), the patient is kept strictly in bed. When work has been assigned, the temperature is watched, and so long as it is not increased by the exertion, the work is increased in duration and intensity. Even afebrile patients who are of poor general condition are not allowed to work, but kept at perfect rest, excepting that they are allowed to walk to and from the dining hall for their meals.

It is thus evident that there is little new in this system of exercises and work. Physicians have always allowed their afebrile patients who are of good general condition and not easily fatigued to work and warned them to stop as soon as symptoms of toxemia, such as a tired feeling, weakness, debility, drowsiness, make their appearance. Intelligent patients have been given thermometers to guide them.

Paterson's method has, however, done a great deal for institutional patients by drawing attention to the importance of exercises and work in attempts at prevention of indolence which, in many cases, remains

as a reminder of the disease and the institutional life to which they have been subjected.

Outdoor Games.—Afebrile patients without tachycardia are to be encouraged to do some exercise in the open air, otherwise they are likely to brood over their troubles, and in some cases even harmed by obesity. Walking exercises alone are often insufficient to keep the average patient busy, and outdoor games are often good to help him pass his time pleasantly and to benefit the muscles, the appetite, and the metabolism.

In advising a patient as to outdoor games we must always consider his life, habits, and customs before he took sick. Those who indulged in sports may be permitted to resume their favorite games, provided they do not raise the temperature, or produce breathlessness. This at once excludes certain games. "All violent sports should be avoided," says Lawrason Brown. "Golf (without the full swing), croquet, fishing and hunting (not entailing too much exercise), gentle bicycle riding (on the level), rowing or paddling, skating (for those proficient), skiing, snow-shoeing, swimming (in great moderation), and horseback riding may be indulged in with moderation when the disease has been arrested."

It seems to me that of the outdoor games, golf is the best for patients who have just recovered from phthisis. Cricket, football, and athletic sports in general produce more or less dyspnea, while golf makes less violent demands on its votaries and is usually played in open, breezy places. However, those who perspire freely are at times greatly harmed by outdoor exercises. "Catching cold" is apt to reactivate quiescent lesions (see p. 125). Though in this bacteriological era many deny the etiological potentialities of colds and chills, experience has taught that they are factors for evil in many cases.

Indoor Games.—The tuberculous patient is to be allowed some games for his amusement when he is kept indoors, excepting when the temperature is above 100° F. and he is kept in bed during the whole day. I believe it is wrong to interfere with them when they play cards, checkers, and chess, as is often done in public sanatoriums, on the assumption that the excitement is liable to raise the temperature, provoke hemoptysis, etc. While it cannot be said that these games immunize patients against such accidents, I have never seen such results follow when they are allowed to have some amusement during the long, lonesome, days and weeks in the institution or at home in the sick room.

Patients treated at home are not to be allowed to go to theaters, or other indoor and badly ventilated places of amusement so long as they have fever.

CHAPTER XXXVII.

OPEN-AIR TREATMENT.

Most writers state that Brehmer was the first to demonstrate, in 1859 in his sanatorium, the great value of the open-air method of treatment of tuberculosis. But it is a fact that he had many precursors. In 1840 George Bodington, a country doctor in the village of Erdington, published an *Essay on the Treatment and Cure of Pulmonary Tuberculosis*, in which he vigorously protested against the close confinement of consumptives for fear of the evil influences of cold, fresh air, "forcing them to breathe over and over again the same foul air contaminated with diseased effluvia of their own persons." Arguing against the value of antimony, calomel, and bleeding, which were in vogue in those days, he urged the free administration of nutritious food and stimulants with plenty of exercise in pure and, if possible, dry, "frosty" air. In short, his great specific in phthisis was dry, cold air which, he said, had a most powerfl influence in "healing and closing of cavities and ulcers of the lungs."

Needless to say, he was severely handled by his contemporaries and so discouraged that he had to give up his method of treatment, converting his "sanatorium" into an insane asylum. Brehmer in Germany and Trudeau in the United States, later took up work along the lines of Bodington and met with no small amount of opposition and ridicule from the contemporary leaders of the profession and the laity.

At present the gospel of fresh air needs no evangelists to bring it home to most sufferers from phthisis; it is the acknowledged cornerstone of phthisiotherapy. The only difference of opinion is where and how it can be applied most effectively. Some send their patients to certain regions where the climate is alleged to have a specific influence on the disease; others direct them to sanatoriums where they may benefit by both the climatic advantages and certain therapeutic methods which are the hobby of the presiding genius. Many are convinced that similar advantages may be obtained at the home of the average patient.

Where Open-air Treatment May be Obtained.—The open-air treatment consists in inducing the patient to live permanently in pure, fresh air, preferably outdoors or, when he must stay indoors, the air in the room is to be renewed constantly. There is no question but that this is best obtained in the country, or in a special institution. But most patients cannot afford to leave the city for an indefinite period, nor are there a sufficient number of institutions in any country to

accommodate all active tuberculous patients with places for as long as the disease lasts. In fact, if all the patients were to decide that they want to submit to hospitalization for therapeutic or prophylactic purposes, it would be found that only a small fraction of the eligible could be accommodated.

Says Edward Cummings:[1] "Personally I cannot see the need of banishing the tuberculous patient from his comfortable chamber to a shack in the back yard, or a woodshed, or a tent house in the dusty desert. One does not always have to go across the continent to get fresh air, not even out in the yard. . . . The ordinary bedroom for most persons is well enough." My own observations in large modern cities like New York, Boston, Chicago, St. Louis, Philadelphia, London, Manchester, etc., have convinced me that results can be, and are, obtained which compare favorably with climatic and institutional treatment. Of course, in the congested districts and slums, the overcrowded tenements are even less suitable for consumptives than they are for human habitation in general. But there are districts in every city which can be utilized for the purpose of housing consumptives and the results attained will not be behind those attained after sending patients far away from their homes at great expense and often hardship.

The suburbs around cities are suitable for families in which there are tuberculous members, and the expense involved in moving them to these parts is comparatively trifling; in fact, the rent is often lower, and they need not lose their jobs, or break up their business. The social and economic aspects of tuberculosis, which are but rarely considered in this connection, assume a different aspect when the patient must not be sent far away from home, or from the place of employment of those he depends on.

Open-air vs. Climatic Treatment.—These two methods must be kept distinctly apart. Experience has taught that there is no climate on the habitable globe in which consumption does not occur, or where a patient sick with the disease will surely recover, even when sent thither in the very incipient stage of the ailment. In the climatic resorts which have become popular—and it is a fact that the popularity of a region is by no means an index of its therapeutic efficacy—the patient must subject himself to a certain discipline, if he expects results. Irrational mode of life in the mountains or at the sea coast will aggravate the condition of a consumptive to the same extent as it will in the city. A healthful mode of life in any place will, and does, improve the condition of the average consumptive, no matter where he is.

The treatment of tuberculosis in certain climatic regions, as we shall see later on, has its indications and contraindications, while home treatment has certain advantages in this regard. It can be applied successfully in the treatment of nearly all cases, in all forms of phthisis,

[1] Journal of Outdoor Life, 1912, **9**, 257.

and in all its stages; striking results are obtained in patients with limited means, as well as in those who are well-to-do; in febrile and afebrile cases; in hemorrhagic and cachectic cases; in those with or without gastric derangements. In short, in all cases of tuberculosis, in all its forms, in all stages of the disease, during any season of the year in almost any climate, except the arid.

To be successful, it must be applied rigorously, methodically, and properly regulated by the physician. This is no more than institutional treatment depends on, excepting that the former is cheaper for the community which is charged with caring for its consumptives, and more attractive to many who have sufficient material means at their command.

Dangers of Stagnant Air.—Our conception of the beneficial effects of indoor life has undergone radical changes during recent years. The reasons why the stagnant air in a room occupied by human beings is harmful are not clear. Recent investigations by Leonard Hill, Haldane, Benedict, Flügge, C.-E. A. Winslow, and others, have shown that it is not the excess of carbon dioxide, or the decrease in the proportion of oxygen which renders the stagnant air harmful. The most deteriorated air in a badly ventilated room never shows on analysis as much as 1 per cent of carbon dioxide, while in famous health resorts at high altitude there is a far greater deficiency of oxygen than can ever be found in the worst ventilated room. The specific organic poisons of human origin, the morbific anthropotoxins, of which some have spoken, have never been isolated.

As Winslow[1] points out, recent studies indicate beyond any reasonable doubt that the more obvious effects experienced in a badly ventilated room are due to the heat and moisture produced by the bodies of the occupants, rather than to the carbon dioxide or other substances given off in the breath. Two fundamental experiments have been repeated again and again which would suffice to demonstrate, as F. S. Lee has so well expressed it, that the problem of ventilation is not chemical but physical—not respiratory, but cutaneous. These are: First, that subjects immured in close chambers and exposed to heat as well as the chemical products formed therein are not at all relieved by breathing pure outdoor air through a tube; and second, that they are completely relieved by keeping the chamber artificially cool without changing the air at all, and are relieved to a considerable extent by the mere cooling effects of an electric fan.

Investigations made by the New York State Commission on Ventilation have shown that the temperature and the pulse-rate of an individual are markedly increased by even a slight increase in the room temperature; they also confirm Leonard Hill's observations that overheated rooms enhance the susceptibility to respiratory diseases owing to changes in the mucous membrane which follow exposure to

[1] Science, N. S., 1915, **41**, 625.

hot and dry air, and the resistance of animals to artificial infection is very definitely lowered by chill following exposure to a hot atmosphere.

In connection with tuberculosis, in which the appetite is of such great importance, it is of interest that stagnant air reduces the desire for food perceptibly. In two series of experiments made by the above-mentioned Commission, standard luncheons were served to the subjects in the experimental chambers and the amount on their plates weighed. In one series the subjects consumed on the stagnant days an average of 1151 calories and on the fresh-air days an average of 1308 calories, an increase of 13 per cent. In a second series during colder weather, the average consumption was larger, 1492 calories for the stagnant and 1620 calories for the fresh-air days.

We have here an explanation for the utility of fresh air in the treatment of tuberculosis. Stagnant air is bad primarily because of its high temperature and lack of cooling air movement, sometimes combined with high humidity. In fact, a lack of humidity, as Phelps has pointed out, makes hot air feel cooler and cold air feel warmer. It is very important that the air in a living room should not be dry, as it is in most of our artificially heated rooms during the winter. Living in stagnant air the patient feels uncomfortable, inert and listless, and above all, loses his appetite for food, which is very essential in the treatment of phthisis. The open-air treatment seeks to remove the drawbacks of indoor life amid stagnant air. For a certain, but limited number of patients it is attained best in a good sanatorium, but it may be just as well attained at home within the city lines in most houses.

Technic of Treatment.—If the patient lives in a capacious home, or in one in which he may have a fair-sized, well-lighted, and ventilated room to himself, in a district or street which is not overcrowded, he may remain where he is. But in case he lives in the slum district of a large city, in a dingy and overcrowded tenement, he must move to better quarters which are available in every city. If his occupation, or that of those he depends on, is not in the way, it is even better that he move to the outskirts of the city, or to a suburb where certain advantages may be obtained which are not available or feasible in the city.

A few words should be said about the various shacks, tents, special window tents, etc., which have been contrived for the city dweller with a view of giving him an opportunity to live outdoors, or in a well-ventilated room. Most of them are not feasible. They cannot be used in the thickly inhabited parts of cities; the tents or shacks cannot be placed in the back yards, on the roofs, etc., without attracting the curious, or even exposing the patient to eviction because of the resentment of the neighbors. I have seen a few patients in New York City who have made use of these contrivances, but they were rare exceptions, and they lived in private dwellings in the outskirts of the city.

But the average bedroom, excepting in the dingy tenements, is sufficient for our purposes. If the patient is allowed to remove the window sashes, both the upper and the lower, as Cummings suggested, he may convert it into open-air sleeping quarters. The patient's room should be large; one with a capacity of 3000 to 3500 cubic feet of air is best. But it must always be remembered that *cubic space is of little value per se unless it is provided with efficient means of ventilation.*

In modern apartments, rooms with windows opening into air shafts, or narrow courts, are not good for tuberculous patients; they should have rooms with windows opening into the street, or a spacious court-yard. In apartment houses with elevators the top floor is the best, the higher the building the better. But in houses without elevators the advantages of the pure air in the upper stories are often negated by the exertion necessary in stair climbing by walking patients; but the ground floor should be avoided in tenements because it is almost invariably dark and badly ventilated. It should also be seen that trees do not obstruct the entry of air and light to the room, and favor excessive humidity. The windows of the room must be located so that the sun's rays enter them for at least part of the day and penetrate at least ten feet into the room.

The walls of the room should be painted, not papered. All unnecessary curtains and hangings should be discarded, leaving nothing but roller shades on the windows. Carpets are obviously bad, but some rugs should be left on the floor. Bare floors are apt to discourage the patient as well as those around him The rugs can be taken out at frequent intervals, aired, and disinfected. The floor should be waxed or painted, so as to be easily cleaned. Steam or hot-water heating is best; gas heating is to be avoided because it consumes oxygen from the air. The usual contrivances to prevent excessive dryness of the air should be used.

Afebrile patients who are allowed outdoor exercises should remain in the room very little during the day. In the city afebrile patients may leave their rooms soon after breakfast and go to some neighboring park where they are to spend the greater part of the day. In the outskirts of the city, or in the suburbs, there may be sufficient space around the house, as well as porches, balconies, etc., on which they may exercise and rest comfortably, read, or do some light work under careful supervision of the physician. Intelligent patients may be given thermometers with directions to guide them as to the effects of exercise or work. Constant use of the thermometer by the patient is, however, a double-edged sword, as has been pointed out elsewhere.

The season of the year has little effect on the outdoor life. The patient is to spend the greater part of the day outdoors during the winter, as well as during the summer. Only intense cold, or sun rays, rain, or strong winds, are to be avoided by seeking shelter. Excepting during blizzards, snow is rather invigorating to the average patient of this class. But here again it is to be borne in mind that very

emaciated and anemic patients, also those over forty years of age, do not tolerate cold.

Sleeping Porches.—Those living in the outskirts of the city or the suburbs may have tents in which they sleep during the night, and seek shelter during inclemencies of the weather. But the usual tent is rather stuffy and damp during the summer and autumn, and too cold during the winter for a tuberculous patient. There are made at present tent houses, or canvas bungalows, which are excellent because of the comforts they afford and the good ventilation that may be had within them.

FIG. 117.—A knitted helmet for protecting head, neck and shoulders. (T. S. Carrington.)

It is, however, best that the patient remain the greater part of the day on the porch and, in most cases, he may sleep in a bed placed on the porch. During the day, in case perfect rest is to be enjoined, he may remain on some form of reclining chair of which there are at present many on the market, such as the Universal Reclining Chair, the Kalamazoo Chair, the common hammock chair, the willow long chair, etc. During the cold winter, if he is not extremely anemic and emaciated and not over forty years of age, the patient may also remain on the porch on one of these chairs during the day, and in a bed during the night. "The whole problem is one of sufficient bedclothes and the use of some sort of hood or head covering (Fig. 117); in short, to dress especially for sleeping out." As Cummings suggests, "by putting on a suit of underwear, a flannel shirt, pajamas of outing flannel, and a hood of flannel or eiderdown, and furnishing the bed with plenty of light weight but warm blankets and comfortables one can sleep with a

continuous flood of fresh air in severe weather with perfect comfort and safety." But it must be emphasized that the number of patients benefited by sleeping outdoors during the winter months is rather limited. Individualization is imperative.

It is self-evident that sleeping porches are only feasible in rural districts, and not in large cities, excepting in their outskirts. But it is always important to remember that the proper construction of a sleep-

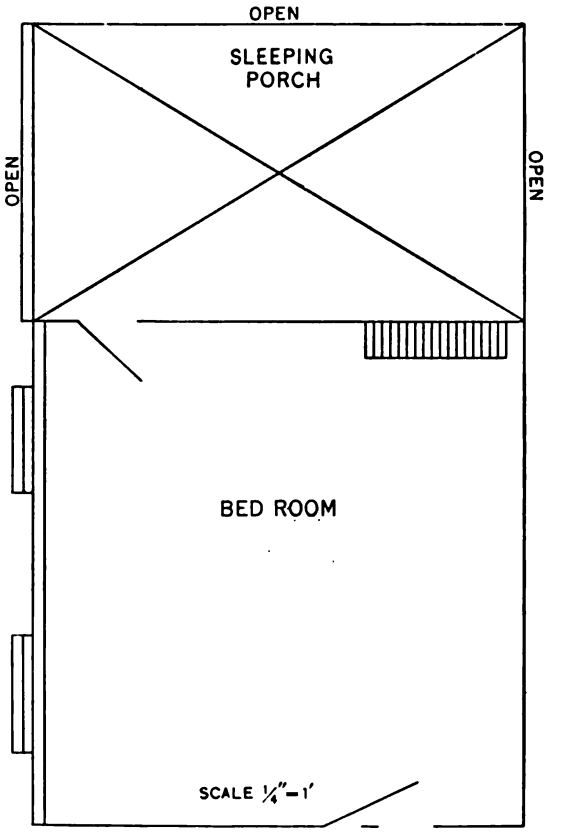

FIG. 118.—Porch exposed on three sides; no provision for keeping the bed warm during the day. (MacWhinnie.)

ing porch is not a simple matter. A. Morgan MacWhinnie[1] investigated 100 sleeping porches in the Northwest and found the following conditions: In 96 cases the sides of the sleeping balcony were partially protected from the wind and rain by a tarpaulin or some other material. Two had no protection whatever, and one was inclosed with glass windows which could be thrown open horizontally

[1] New York Med. Jour., 1914, **99**, 780.

at night on retiring. This was the only one that could be closed in the daytime, and had hot-water radiators connecting with the boiler in the cellar that kept the bed and its coverings as warm all day as the rest of the house. In 98 cases the bed, mattresses, linen, and covers were exposed all day to the dampness of the atmosphere. I found similar conditions in most of the sleeping porches in the East.

The warming of the bedding and coverings and keeping them dry are elements which are very often neglected in open-air treatment, and it is not surprising that most patients cannot sleep outdoors on cold

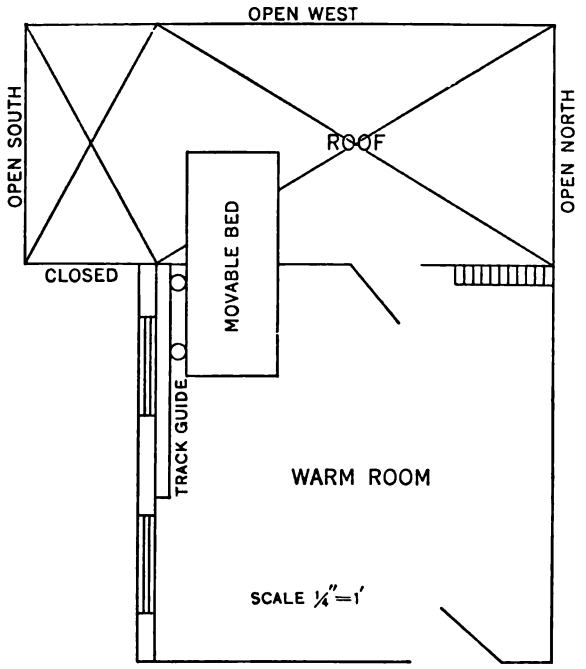

FIG. 119.—Ideal sleeping porch. When the bed is fully extended on the porch, the footboard closes the room from the outside air; when bed is in warm room, headboard closes opening to sleeping porch. (MacWhinnie.)

and moist days. Moreover, an attendant must be engaged to attend to these details, and this is costly for the average patient. MacWhinnie suggested sleeping porches which have none of these disadvantages; they are so arranged as to be completely protected from the weather. He urges that the doors should be large so that the bed can be kept in the heated room during the entire day and bedding remains warm and dry. When ready for the night, it should be wheeled to the sleeping porch, thus obviating disadvantageous conditions mentioned above.

Figs. 118 and 119 show the plan of a sleeping porch, designed and constructed by Dr. D. C. Hall. An opening is made in the wall large

enough for the bed to roll through to the porch. The head and foot boards are so constructed that the opening in the wall is entirely closed when the bed is at full length on the porch or in the room. The room is thus kept warm for dressing in the morning. The bed is supported by four large roller-bearing wheels, one hand of a child sufficing to move it out or in. Grips are so arranged that the bed can be drawn out or in, while the occupant is in the reclining position.

The afebrile patient may indulge in driving, automobiling, or sleighing during the winter, but always within the limits set by the physician.

He should discard many of the pleasures of healthy people, even when he thinks he is well; he should not visit theatres, balls, crowded restaurants, etc., where large numbers of persons congregate and contaminate the air. Many a patient who has been doing well, and on the road to recovery, has suffered a relapse, or a complication, after attending a function at which a large number of persons got together in a confined space. Many suffer relapse, or reactivation of the disease after exposure to the vicissitudes of the weather.

Open-air Treatment of Febrile Patients.—With febrile cases things are not so simple. They must remain in bed as long as the fever lasts, excepting under circumstances which are discussed elsewhere. In the city, the bed can only be kept within the room, and for this reason, as well as for others, it must be placed near the window, so that not only pure, fresh air may be avilable at all times, but also because the patient is usually encouraged looking out at the living world. In the suburbs the bed may be placed on the porch during the day during the summer months, and under certain circumstances it may remain there all the time. When feasible, a proper tent or porch is even better. Placing tents on roofs of houses in the city, or modifying fire escapes so that the patient may be kept on them in the open air, is not feasible. No patient wishes to expose himself to the curious gaze and commiseration of the other inhabitants of the house, as was already mentioned.

The good effects of the open-air treatment are often very striking in febrile cases. The general condition of the patient improves, a feeling of well-being ensues, replacing the despondency into which he was sinking. His strength returns. The anorexia and indigestion which sapped his strength disappear, or are ameliorated, and he eats with a better appetite. The painful cough often disappears within a few days and nights with open windows, or on the porch. This is at times the most salutary phenomenon; sometimes when sedatives have failed to control the cough, outdoor life works in this direction and the effect on the morale of the patient is marvellous.

During the night open-air treatment is even more simple than during the day. It consists in one principle—open windows. There is no room in a decent house which cannot be properly ventilated by opening the windows, excepting during some of the hot and humid summer months in certain regions. But then the conditions outdoors are not much superior, and a tuberculous patient should not remain

during the hot and humid summer months in his home. If he cannot be moved, an electric fan will renew the air in the room properly.

When dealing with young and plethoric individuals, and those who have been adapted to open air life, the windows may be opened completely; the upper half may be lowered, and the opening should not be obstructed by any shade or curtain. But during the winter heat must be supplied in all cases, though the windows may be lowered after the patient has been properly covered. A sufficient number of woolen blankets, and plenty of flannel underwear, should be available for use, as required according to the temperature. There are people who can dress and undress in cold rooms, but they are rare; and tuberculous patients, especially those who are anemic and cachectic, cannot stand cold with impunity. But proper ventilation may be maintained in a room which is properly heated.

Abuses of Open-air Treatment.—In an effort to get away from the stuffy wards and rooms in which tuberculous patients were formerly kept, we have recently gone to the other extreme and attempted to subject all patients indiscriminately to the open air treatment during all seasons. But it has been observed that a large proportion is not fit to live outdoors during the winter months. There are many healthy, as well as tuberculous, whose constitutions, or even habits which have been theirs since birth, are not adapted to cold air, and they, at times, suffer severely when persisting in carrying out the treatment as outlined above. I have seen many patients distinctly harmed by life in a cold atmosphere. There are persons in whom cold induces catarrhal conditions of the respiratory passages, and even of the intestines; indiscreet exposure to cold is at times responsible for pleurisy, bronchopneumonia, etc. Insomnia in the tuberculous is also very often the result of living in a cold room. Healthy persons are often kept awake in cold bedrooms. It has been found that in moderate climates, tuberculous patients feel better during the winter. But vital statistics show clearly that the mortality from this disease is highest during the winter and spring months, indicating that complications are more likely to arise during the cold months. There is no doubt that complicating bronchopneumonia, pleurisy, and certain forms of bronchitis, which are common in tuberculous patients during the cold months, and which are often the last straw, could be prevented in many cases by avoiding exposure to cold.

Open air treatment has also been abused while attempting to "harden" tuberculous patients. In many children the process of hardening, cold baths, frictions, and exercises, are beneficial. But in adults it is just as often harmful, especially when they suffer from tuberculosis. As has recently been pointed out by Köhler,[1] open air life is beneficial because it stimulates the production of heat in the body during the reaction which it induces; but these benefits are

[1] Ztschr. f. Tuberkulose, 1920, **31**, 321.

observed mainly in persons before the period of katastasis, *i. e.*, before thirty-five years of age. Katastasis occurs in most tuberculous patients much earlier than thirty-five years, and for this reason it is dangerous for many of them to persist in living in a cold atmosphere. While pure air is usually cool, it must not necessarily be intensely cold; and renewing the air in a room should not involve the production of draughts.

To keep an anemic and cachectic tuberculous patient comfortable, heat must be applied, especially to the extremities. Nearly all tuberculous patients over forty years of age, and a large proportion of younger ones, are safer in a warm, but well-ventilated home during the cold or chilly months of the year. All tuberculous patients should be properly clothed according to the season, and woolen underwear is imperative during the winter in most cases. Cold baths, taken in cold rooms, are decidedly dangerous for tuberculous patients, and for this reason it is, at times, impossible to find proper accommodations for them in the country during the winter. I have been convinced that many patients have been harmed while attempting to live in the country during the winter in houses and shacks which were not properly heated. In their homes in the city they can easily have pure and circulating air which is cool, but not cold. Of course, many patients have been "hardened" in this regard, but a large proportion succumb during the process.

While attempting to ventilate sick rooms during the winter, draughts are to be avoided for the above mentioned reasons. Sudden changes in the temperature of the room should be prevented unless the patient is well covered. When leaving a warm room into the open air, appropriate clothing is to be worn; many patients begin to cough soon after they emerge into cold air. Driving in open vehicles during cold and windy days should not be indulged in by tuberculous patients. Many complications, especially pleurisy, bronchitis, and catarrhal enteritis, may thus be prevented.

It has been the universal experience that when the summer heat is accompanied by excessive humidity, tuberculous patients suffer from anorexia, insomnia, general weakness, etc., and they often lose the greater part of their gains during the winter. For this reason I insist that all patients under home treatment should leave for at least July and August for the mountains. But during the winter the majority of tuberculous patients are better off in the city, unless they are kept in the country in houses or institutions which are properly heated.

Results Attained by Open-air Treatment.—The results attained by the open-air treatment depend on many conditions, notably the acuteness and the stage of the disease. In acute, progressive cases we cannot expect much more than from any other method of treatment, excepting perhaps more comfort to the patient than would be the case if he were kept indoors. The ultimate prognosis is gloomy at all events.

In subacute cases the process is at times arrested and the disease then pursues the course of chronic phthisis.

The good effects of the open-air treatment are best seen in the average case of incipient chronic phthisis which begins with moderate fever, nightsweats, anorexia, cough, etc. In advanced cases of the disease, when the patient is emaciated and apparently hopeless, several days of life in the open air often transform a despondent individual into one who shows his confidence in ultimate recovery very clearly. He gains in courage and is imbued with a desire for recovery; his fever declines, the nightsweats disappear, the cough and expectoration diminish, and he becomes hopeful in general.

In the far-advanced stages of the disease the open-air treatment may only render the last days of life somewhat more bearable, contribute to the false optimism which is often seen in these patients, and accentuate the euphoria which has been considered characteristic of the disease. But it is undoubtedly curative in the vast majority of incipient cases. The entire aspect of the patient is often transformed within a week or two, and the improvement is usually progressive. A good appetite with proper assimilation and digestion of the food, disappearance of the fever, nightsweats, insomnia, and amelioration of the cough, are the rule in these cases. Often it will be noted that fever, which resisted all other treatment for months, disappears after several days of life with open windows during day and night. Many patients learn it by experience and cannot be induced to close the windows. They have found that with open windows they sleep better, and feel refreshed in the morning, while closed windows induce cough, nightsweats, insomnia, listlessness, etc.

Contraindications.—It must be emphasized that there are but few contraindications to the open-air treatment. Even hemoptysis, however severe, should not induce us to close the windows of the room inhabited by a tuberculous patient. Nor should they be closed during any season, provided the room is properly heated, as was already mentioned. Only during the summer, when the external air is often hot and humid, and even open windows are not effective in producing a free circulation of the air within the room, this method is often futile. An electric fan may improve conditions somewhat, but it is best that patients who can afford it should leave the city for a milder or colder region.

There are many patients who do not bear the open-air treatment very well during the winter months; in fact, in some it is distinctly harmful, and if an attempt is made to apply it, it must be done with great care and circumspection. Patients who suffer from diffuse bronchitis in addition to phthisis do not bear cold air very well and so-called "rheumatic pains" in the joints are often aggravated by sleeping in a cold room; also those in whom the heart has been profoundly affected by the tuberculous toxemia—the poor circulation results in acrocyanosis, cold extremities, etc.; dyspnea and the cough are decid-

edly provoked by winds, draughts, and cold air in general. Those suffering from profound anemia at times cannot be kept warm by any means in a cold room. The same is true of old persons with bad peripheral circulation and extremely cachectic patients—they cannot be kept comfortable in cold rooms during winter nights.

In all these cases it is necessary to heat the room, but the windows should under no conditions be closed completely.

Phototherapy.—The value of light, especially sunlight, as a therapeutic agent in tuberculosis has been appreciated for centuries. Climatic resorts, extolling their therapeutic efficacy, almost invariably mention the number of sunny days in the regions they are located. More recently Rollier[1] has written considerably about the great success he has attained in the treatment of articular, osseous and glandular tuberculosis in children by exposing them to direct sunlight in a certain fashion. Numerous other authors report excellent results not only in tuberculosis of bones and joints, but also the pulmonary forms of the disease.

We have shown elsewhere that in extrathoracic tuberculosis any harmless drug, or therapeutic procedure, will give good statistical results, because the vast majority of cases tend to recovery, as long as they are kept at rest and not harmed by irrational surgery and medication. Likewise, when exposure to the rays of the sun is cautious, no harm results in most cases of incipient, mildly active, and even advance disease which shows tendencies to sclerosis of the lesion. The statistical results may even appear excellent because of the salutary psychic effect, the patient feeling that something is being done for him. But when sunbaths, and other modes of application of direct sunlight are attempted in active and progressive pulmonary tuberculosis, and in laryngeal disease with especially contrived mirrors, the results obtained are not very striking. Indeed, when practised indiscriminately, deleterious effects are observed: Superficial burns, and even focal reactions in the lungs, contributing to extension of the tuberculous process, may occur.

More recently "artificial sunlight" has been lauded, especially by the manufacturers of certain lamps which have been urged on the profession, and incidentally to tuberculous patients, at an exorbitant price. Having obtained good results in the treatment of certain tuberculous skin lesions by the use of the carbon arc light, Finsen urged the violet and ultraviolet rays of the spectrum as a therapeutic agent in treatment of pulmonary tuberculosis. Later, however, the mercury quartz lamp, of which there are now several models on the market, displaced all others.[2] This mercury quartz lamp consists of a quartz container supporting mercury in a vacuum through which an electric current is

[1] La cure de Soleil, Paris, 1914.

[2] For a very thorough review of the literature and therapeutic results of sunlight and artificial light in the treatment of tuberculosis, see Edgar Mayer, Am. Rev. of Tuberculosis, 1921, **5**, 75–158.

passed. It is noteworthy that this lamp does not produce anything comparable to sunlight; the heat rays are almost entirely lacking, while the violet and ultraviolet rays are excessive at the expense of all other rays.

It appears that here also inactive tuberculous lesions in the lungs are said to be benefited by the application of these violet and ultraviolet rays. Some have reported good results in cases of intestinal ulceration complicating pulmonary tuberculosis, but others have failed to obtain them. The same may be said about the application of these rays in laryngeal tuberculosis. From Mayer's digest of the literature, and his own experience, it appears that in active and progressive tuberculosis, the quartz lamp has not shown any value as a curative agent. When applied with caution, there is one thing in its favor—it is a harmless mode of treatment. But when abused, it may cause headache, insomnia, tachycardia, and reactivation of the tuberculous process in the lungs, with their concomitant symptoms and results. Burns of various degress are not uncommon when the quartz lamp is recklessly applied, and at times large blebs result from over exposure of persons with delicate skin. However, from personal experience, as well from the reports of even overenthusiastic advocates, it appears that phototherapy can hardly be credited with any therapeutic value in tuberculosis in any of its forms.

The same may be said of the treatment of this disease with the roentgen rays. Many authors, notably Bacmeister, Kupferle, De la Camp, and others, have reported good results when these rays are properly and cautiously applied according to a certain technic which they have worked out. But when we find here also that only afebrile and inactive cases are benefited, our suspicions are aroused that when used in small doses they may exert a favorable influence because the patient feels that something is being done for him; when used in large doses, by those who have not mastered the technic of roentgen therapy, harm may result of even greater magnitude than that lurking in phototherapy.

CHAPTER XXXVIII.

CLIMATIC TREATMENT.

We have seen that the vast majority of tuberculous patients are amenable to home treatment; if they are to recover at all, they can accomplish it without leaving their home surroundings. The autopsy findings showing that many persons have healed tuberculous lesions in the lungs and pleura, although they have never undergone a course of institutional or climatic treatment, prove clearly that tuberculosis is curable in all climates. But there are undoubtedly indications for certain forms of climatic treatment in tuberculosis, though they are not as imperative nor as necessary for the average case as the laity and part of the profession believe. In this chapter we shall attempt to review the indications and point out the limitations of climatic treatment.

Climatic treatment of tuberculosis is probably older than any other method which has survived the recent advent of scientific medicine. The ancient Greek and Roman, as well as the medieval Arabic physicians were great believers in the efficacy of certain climates in the control and treatment of phthisis. The first thought that enters the mind of the average modern physician after diagnosticating a case of tuberculosis is, "Where should I send the patient?" If the physician is negligent in this regard, the patient will surely ask him, "Must I leave the city?"

It is, however, a fact, agreed to by all entitled to an opinion, that *recent studies of the effects of various climates on the incidence and the course of phthisis have not resulted in discovering a region on the habitable globe which can be relied on to cure or improve all incipient or a substantial proportion of advanced cases of the disease.* Whenever geographical, topographical, meteorological, and clinical data are correlated with demographic data for a given locality, and conclusions drawn that a very high percentage of cases recover while living there, there are at once shown other facts which prove conclusively that under climatic conditions diametrically opposed to these, the proportion of recoveries is about the same. For these reasons many physicians have gone to the opposite extreme and claim that climate need not at all be considered as a therapeutic agent in the control and cure of phthisis.

Economic Aspects of Climatic Treatment.—Other reasons militating against the extensive utilization of certain climates may be mentioned. Bearing in mind that the bulk of consumptives are recruited from the poorer strata of society and that even those who had been self-sup-

porting before they were attacked by the disease often become dependent soon after that event, it is evident that the economic factor is to be given great weight in this connection. Indeed, climatic treatment is as expensive as institutional treatment; it is even more beyond the reach of most patients because modern municipalities provide, as a rule, institutions for the tuberculous, but hardly any supply funds with which patients may go to distant parts of the country and support themselves for a considerable time.

This economic aspect of climatic treatment is too often disregarded by physicians who tell their patients, irrespective of their financial condition, to go to distant regions. Those who cannot raise the funds and must stay at home become despondent, and the prognosis is often aggravated as a result of it. Some of them go with meager funds to Colorado, Arizona, California, etc., and the result is even more disastrous.

Cost of Climatic Treatment.—Thompson Fraser,[1] who has made a study of this problem in Asheville, N. C., and reported his observations in the Public Health Reports, shows that it must always be borne in mind that there is a clear relation between income and recovery in tuberculosis. When leaving for some climatic region, the patient must be prepared to provide himself with the proper requisites. If he lacks funds he should not undertake a trip which not only exhausts his resources, but does him no good; he should rather stay at home. He points out that at Asheville, and this holds good for nearly every other climatic resort in this country, the expense is about as follows:

The cost of room and board varies within wide limits. From his observations at Asheville, board of fair quality with room costs from $10 to $12 a week at the houses which are licensed to take tuberculous patients. The price depends to some extent on the location of the rooms, the more desirable ones costing more, while less desirable rooms may be had for $8. The "extras," Fraser points out, amount to almost as much as the cost of the room and board, including, as they do, additional food, milk, eggs, reclining chair, physicians' fees, medicines, thermometers, blankets for cold weather, laundry, and everything that comes under the item of "incidentals."

Fraser's conclusions are that the cost to the patient for a period of ten months, or forty-three weeks, at $8, $10, $12, a week would be $344, $430, $526, respectively, not including the extras just mentioned. A minimum of $700, therefore, exclusive of car fare, would be a more just estimate of the expense for the rather arbitrary period of ten months. These calculations were based on costs before the war. At present more than double the sums mentioned are necessary. If the patient is accompanied by some member of the family, it may be decided to keep house instead of to board, but this will not prove more economical in most cases.

[1] Public Health Reports, September 18, 1914, 29.

The estimate for room, board, and treatment for a period of ten months applies especially to those cases which can be benefited by a comparatively brief stay. If the disease has made greater inroads, and a longer stay is necessary to produce results, the cost of extras and perhaps of nursing may be prohibitive to the average consumptive, and it is wiser to remain at home where suitable food, care, and comforts will more than outweigh the benefits of climatic factors, if unassisted by these essentials.

Climatic treatment is thus a luxury available for the chosen few, while the vast majority of sufferers from tuberculosis must perforce remain in their homes for treatment.

Whenever I decide in favor of climatic treatment I inquire whether the patient has resources to hold out for at least one year. Under present conditions I insist that the expense will be at least thirty dollars a week. If he cannot raise that amount I discourage him from leaving the city.

Effects of Change of Environment.—Looking with a sane and unbiased view on the problems of climatic treatment of phthisis, we find that it is undoubtedly an important adjuvant to our efforts at curing our patients. Even physicians who practice in cities, and have good results with home treatment are often impressed with the salutary effects of a change of surroundings. One has but to note the effects on a patient who has been kept at home for several months and all available hygienic, dietetic, and therapeutic measures to control the disease have been taken, yet the patient has been going steadily downward. A change in surroundings is decided upon, and he is sent out to the country, preferably a place the patient selects, provided there are no strong objections to it. It makes no difference whether the locality selected is at the sea coast or inland, in a forest or a desert, on a high altitude or the plains; it is immaterial whether the number of sunny days calculated by the weather man, or by the owner of the resort in the neighborhood, is small or large, whether it is foggy or even frequently rainy—the results are often astonishing. After remaining there for a few months, the patient returns greatly improved, in some cases even apparently cured. These are the facts which every observing physician is bound to meet in his daily practice and cannot be controverted by statistics or opinions of famous clinicians. But it is clear that in such cases it is not the meteorological or topographical conditions which are altogether responsible for the good results attained by the change.

Carefully analyzing the results obtained by patients under my observation, I have arrived at the conclusion that the complex phenomena grouped under the title "change of environment," or the psychic and biological response of the organism to a change in surroundings, play here a greater rôle than the difference in the composition and density of the air, or the number of sunny and foggy days. The change in environment acts as a new stimulus, reinvigorates, and calls forth the dormant vital forces of the patient.

Suggestion is a factor in climatic treatment of tuberculosis which has not been given the credit it deserves. The patient has heard that a consumptive cannot recover in the city, and, when unable to leave for any reason for some place reputed to be efficacious in this direction, he becomes despondent. Many brood over it to an extent as to nullify all other therapeutic measures. Once they are sent away, all potential and inherent vital forces are stimulated; despondency is replaced by a feeling of hopefulness, accompanied by an increase in the appetite, improved assimilation of food, diminution in the cough, etc. This is proven by the following facts which have come under our observation:

Patients leave their homes where they have been under the tender care of relatives and have had good and properly prepared food, and go to the mountains or the sea coast where they are compelled to live in cheap boarding houses or hotels, in which the food given them is far inferior to that which they had been getting at home. Yet they thrive and gain in weight, while at home they had been wasting progressively. Others go to hotels and boarding houses which, for obvious reasons, allege in their advertisements that, in the reality much-coveted, consumptives are barred. In fear that when coughing the proprietor of the hostelry is liable to discover their ailment, the patients promptly cease coughing. In many cases the gain is only temporary, and after the so-called acclimatization, the "climate wears out." But the gain is immense in a large proportion of cases. The disease often takes a turn to the better, or the patient is carried over an acute exacerbation and given an opportunity to recover his inherent vital forces.

This effect of a change of environment is often seen in patients, themselves natives or residents of agricultural districts, even high mountainous regions, who have become sick with tuberculosis, and coming to the city to consult a physician improve, in spite of the fact that climatic conditions are undoubtedly inferior. But there has been a change of environment.

That it is not entirely the climate *per se* which is responsible in all cases which improve by a change, is acknowledged by most authorities on medical climatology. Henry Sewall[1] points out an antagonism between the vital effects immediately attendant on a change of climate and those, often totally different in character, which may develop during permanent residence. "In short, a change of scene, irrespective of the character of the environment, has often temporarily a mysterious influence for good on the living organism. The first vital reactions to new climatic conditions involve especially the nervous system, the final effects are dependent on the modified metabolism of the individual organs, and this may or may not be conducive to the efficiency of the body as a whole." Brown puts it pointedly when he says that without doubt many of the effects attributed to climate can be ascribed to change of climate.

[1] Klebs' Tuberculosis, p. 664.

The writer has observed patients who left a favorable climate, where they have done badly, for an unfavorable one, where they soon improve wonderfully. Many immigrants who become tuberculous in New York City, try institutional treatment and fail to improve. A longing for their native land overtakes them, and they leave for home where they remain for some months and return to this country cured. We have observed numerous instances of this kind in New York. From personal observations, the writer can testify that the hygienic, sanitary, economic, and social conditions in southern Italy, Hungary, Russia, and Poland, where these patients go, are inferior to those in which they live in New York. Indeed, tuberculosis in those countries is more ravaging than here; is more often fatal. Nor are there sufficient accommodations for dependent consumptives. Still, many immigrant patients, who fail to get relief in the many excellent public sanatoriums in this country, in the mountainous regions of Colorado and Arizona, or the beautiful parts of Southern California, go to some large or small city in southern or eastern Europe and, after remaining there for several months, return apparently cured and able to work.

There is no doubt that in such cases it is not the climatic conditions that helped, but the confidence they placed in their native lands, in the home surroundings, in the caressing tenderness of loving relatives, etc., which was instrumental in awakening the reparative forces of the organism.

There are other reasons for sending patients, who can afford to go, to some region with a favorable climate. It is very often difficult to enjoin complete rest and freedom from the worries and anxieties of every-day life in the home of the patient. Nor can he be kept from the temptations of city life. These objects may be accomplished by removing him from his home environment into some secluded country place. The patient is to be told that he will have to remain away from home for several months and he should not leave unless he has sufficient funds for the purpose. His relatives are to be warned against informing the patient of any troubles at home. To this must be added the regular hours for meals, rest, exercise, etc., which are followed implicitly in the country, but often disregarded in the city with its temptations. I have had results which were astonishing with patients sent away in this manner.

With some patients institutional treatment is best for these reasons, as will be shown later on, while with others the reverse is true. In fact, many patients are better off when sent out to roam freely in the country than when sent to closed institutions.

Where to Send Patients.—Experience has shown that for the vast majority of cases of incipient and uncomplicated phthisis it makes little difference whether they go to a mountainous region or to lowland, to the sea coast or inland, to a moderate or cold region; the effect is practically the same, as long as they are taken away from their homes and placed under favorable surroundings, away from the troubles of

home life. There is no climate which cures consumption, the many laudatory advertisements of institutions and railroad companies notwithstanding. The fact that nearly all successful sanatoriums, located as they have been in such a diversity of climatic environments, show practically the same proportion of cured, arrested, improved and last, but always least, dead, proves conclusively that if the climatic conditions are a factor, they are of least importance.

A careful perusal of Guy Hinsdale's prize essay on *Atmospheric Air in Relation to Tuberculosis*, which is one of the best books on the subject, and most impartial, because the author is not anxious to "boost" some region or institution, shows clearly that climate is of little therapeutic importance in tuberculosis. He admits that good results are obtained in cloudy regions, as, for instance, in the Adirondacks, and at Rutland, Mass. He has no objection to sunshine, because the moral effects of bright sunny days, and plenty of them, are very great. As to the question of temperature and humidity, Hinsdale concludes that the majority of incipient cases do best in dry and cool places "not warm enough to be relaxing, but not so cold as to be repellant and restrict exercise and out-of-door life." The old ideas about equality of temperature, at least between the temperature of midday and midnight, are not of great importance; all mountainous stations show great variations in this respect. Some variability tends to stimulate the vital activities, but in older people and those who are feeble, great variability is a disadvantage. Hinsdale denies that altitude *per se* has any great influence. It is of benefit mainly because it is incidentally associated with mountain life, with more sun, less moisture, and scattered population. One statement made by this author should be reprinted with heavy type in all discussions on the subject. "That a place is frequented by consumptives does not prove that it is a desirable place for them."

Mountain Climates.—When a change has been decided upon, the first thought which enters the mind of the patient, as well as that of the physician, is whether a high altitude is best. High climates have been popular for centuries; even ancient physicians, who believed that phthisis is invariably fatal, sent their patients to the mountains when feasible. Most of the modern sanatoriums are located in regions of more or less high altitude.

We do not know why high climates are beneficial for consumptives. Various hypotheses have been formulated to explain it, but none have been proven. The purity of the air is beyond question; the absence of massed population assures freedom from air contamination. Humidity is also less frequent, though not so rare as some would lead us to believe, and many sanatoriums are located in regions which are notorious in this regard. The air is cool, even during the summer, especially in regions of 4000 feet or more above sea level. But the cold is not felt as acutely even during the winter owing to the greater diathermancy. The ozone, of which many writers of past generations spoke so much,

has been found to be worthless. There is very little ozone, and even if there were more we do not know that it would do much good to the patients.

The diminished atmospheric pressure and rarified air have been considered beneficial by increasing the mobility and expansibility of the thorax. It promotes deeper, fuller, and more frequent respiration. But how much of this is due to the outdoor life and whether outdoor life at lower altitudes has not a similar effect on consumptives, have never been satisfactorily investigated.

The effects of high altitude on the hematopoietic organs and tissues have been investigated and some have found an increase in the amount of hemoglobin, others, a polycythemia, still others an increase in the number of leukocytes, etc. Webb and Williams[1] have found an increase in the lymphocytes, or mononuclear elements of the blood, as an effect of high altitude. Some authors, notably Bartel, Bergel, Marie, and Fliessinger, have seen in this increased lymphocytosis in tuberculosis a defensive attempt on the part of these blood cells, while others see in it a demonstration that the lymphocytes contain a lipolytic ferment which destroys the waxy coat of the tubercle bacillus. Minnie E. Staines, T. L. James, and Carolyn Rosenberg[2] confirmed these findings in Colorado. They found that at an elevation of 6000 feet the larger lymphocytes are absolutely increased in the circulating blood by at least 20 to 30 per cent in both man and monkeys. Webb, Gilbert, and Havens[3] found an increase in the blood platelets in tuberculous human beings and monkeys, and that at higher altitudes the increase is even more pronounced. But that these blood platelets contain or supply opsonins, or that they play a rôle in the cure of tuberculosis has not been proved. On the whole, it appears that the hematologic studies of phthisical subjects are contradictory and it has been shown that the conflicting findings have been due in a great measure to errors in technic. It may be stated that the hypotheses promulgated by some authors have not been confirmed by facts observed by other investigators.

Some have maintained that the proliferation of connective tissue in the lungs, the true reparative process in phthisis, is enhanced by a residence in the mountains. But von Muralt, who formulated this theory, has not given any substantial and convincing proof.

Even the statistics tending to show that deaths due to tuberculosis are less frequent in mountainous than in other climates have not withstood scientific tests. It appears that tuberculosis was rare in the Rockies, the Andes, etc., so long as the population was sparse, the inhabitants leading an outdoor life, etc. But since cities have been established at high altitudes and social conditions favoring the development of phthisis created, the disease is not infrequent among the

[1] Tr. Nat. Assn. Study and Prevent. Tuberc., 1909, **5**, 231.
[2] Arch. Int. Med., 1914, **14**, 376.
[3] Ibid., 1914, **14**, 742.

indigenous population. The American Indians, when infected with tubercle, succumb to the disease despite residence in the mountains.

It is thus clear that economic and social conditions play the same role in the cure of tuberculosis in the mountains as they do in the plains or at the sea coast. On this point all authors are agreed. *When a patient goes to a high climate, penniless, and starves there, he will succumb just as quickly as he does in the slums of the city. If he works in Phœnix, Denver, etc., while the disease is active, he may breathe all the rarefied air, expand his chest to an extreme degree, and still succumb just as quickly as in the city.* It is only those who can afford rest, good nourishment, and careful medical supervision who are benefited by life in a high altitude, and most of these are also doing well in other climates.

Indications for High Climates.—High climates are no panacea for tuberculosis; in some cases they are not an unmixed blessing. They have their indications and contraindications.

Patients in whom a positive diagnosis of active phthisis cannot be made but who nevertheless show symptoms and signs of the disease—in other words, the so-called "suspects"—may be sent to the mountains for a short or long stay on the principle that they need a rest anyway. But we must be careful and not suggest such a vacation to those with limited means. I have seen self-supporting artisans ruined, their children committed to asylums, while the father was sent away to the mountains without a positive diagnosis of tuberculosis. That they returned within a month or two reinvigorated and in excellent health was not sufficient to justify the sacrifice; the same result could have been obtained by less costly means. It is different with the well-to-do, who mostly court a vacation.

A large number of neurotics, anemic and debilitated individuals who are in constant fear of tuberculosis, and in whom a diagnosis has been made by some physician, but careful examination fails to elicit any symptoms and signs pointing to a lesion in the lung, are nearly always benefited by a stay in the mountains. Phthisiophobia, which may be considered a distinct syndrome common in modern times, should be treated in the mountains when patients can afford the change. They may remain under the impression that they have been cured of tuberculosis, but this does not make any material difference so long as they are relieved.

Many of these "suspects" and "phthisiophobiacs" may have been cases of abortive tuberculosis in which the physical signs were indefinite or absent. The rest in the mountains and the change of environment undoubtedly contribute to their recovery.

Incipient cases of tuberculosis with few constitutional symptoms gain considerably by a change for a mountainous climate. The appetite improves, the anemia vanishes, and they often gain in weight better than they would have in the city with its temptations. The patients are also freed from the troublesome solicitations of their

relatives and friends which are often more a detriment than a help to recovery.

Active phthisis in the moderately advanced stage which does not improve under home treatment for any reason may be sent to the mountains for a prolonged stay. It is at times surprising to see marked improvement manifesting itself soon after their arrival in the country. Fever is no contraindication, provided it is not of the hectic or terminal variety, or due to some complication which may be aggravated in a high altitude. *Occasionally a pleural effusion showing no tendency to absorption will disappear after a stay in the mountains.* F. L. Knight preferred patients of phlegmatic temperament to the nervous, with irritable heart, frequent pulse, and inability to resist cold.

Of course, most tuberculous patients who can afford the expense should be sent to the country, preferably the mountains, during the hot and humid summer months.

Contraindications.—As was already stated, high climates are like a double-edged sword and may be harmful. As a general rule it may be said that *hopeless cases, running an acute course with hectic or high continuous fever, with a rapid extension of the process in the lungs, profound emaciation, edema of the extremities, etc., should not be sent, for obvious reasons.* It is a great pity to send them travelling great distances, which aggravates their already bad condition, to suffer or die among strangers. Their relatives are also to be considered. Upon hearing of the desperate condition of the patient on his arrival at his destination they may have to go to see him.

Some of these progressive and apparently hopeless cases take a turn for the better with careful home treatment; the fever abates, the appetite improves, the strength begins to return. At this stage it may be well to send them away to the mountains where the improvement which began in the city is enhanced by the new surroundings. At any rate, they do not lose by the change and, when they can afford it, it may contribute greatly to their ult mate recovery. But they need experienced nurses to take care of them.

Dyspnea is a strong contraindication to a mountainous climate. It is often not considered and the results are disastrous. Consumptives with dyspnea due to pulmonary emphysema, asthma, and fibroid phthisis, all of which mean cardiac dilatation; or due to cardiac hypertrophy of a high grade, fatty degeneration of the heart muscle, nephritis, arteriosclerosis, etc., should not be sent to a high altitude. F. L. Knight objects to persons over fifty years of age. Tachycardia, when the pulse is much over 100 per minute, and not slowing down after a long rest, is also a strong contraindication.

Amyloid degeneration of visceral organs, advanced laryngeal, intestinal and peritoneal tuberculosis are contraindications. This is not because the climate is harmful, but the hopelessness of the case precludes sending the patient far away from home. Schröder, whose experience has been very large, warns against sending patients with signs of com-

mencing cardiac weakness and with strongly accentuated neuroses to an altitude of over 1000 meters above sea level.

In selecting a patient for a high altitude, we must not put very much weight on the climatic action on the pulmonary lesion; it is its influence on the heart, bloodvessels, and nervous system that is important. If distinct disturbances in the structure or function of these organs are found, we must warn the patient against high climates. If there are strong reasons for sending him there, it must be done slowly—sending him first to a medium altitude and watching the effect, and when no harm is done he may be permitted to go higher, and finally, if he bears it well, he may go up as high as 6000 feet or more above sea level. It is obvious that these experiments can only be made with economically independent patients.

It has been repeatedly stated that hemoptysis is more likely to occur in high altitudes than on the plains, but this is not substantiated by facts observed by physicians with extensive experience in the mountains. *Available evidence tends to show that pulmonary hemorrhages are no more frequent on mountains of moderate height (2000 to 5000 feet) than in lower regions.* Some authors, like Turban, state that they are even less frequent.

The writer has sent to the mountains many patients with strong proclivities to bleed while in the city, and with the improvement in the general and local conditions, the tendencies to hemoptysis also disappeared. I have often been shocked by the advice given to patients who happen to get a hemorrhage while sojourning in the mountains, to leave at once, and they are in fact taken, while still bleeding, on a long journey. Moribund patients are thus brought to the city occasionally.

Hemoptysis may occur in the mountains as well as in lower regions; it has not been proven that it occurs more frequently in the former places than in the latter. It seems, however, that the results of a copious hemorrhage may be more often serious in the mountains, especially in patients with impaired circulations, as has been shown by F. C. Smith.[1] His statistics show 56 deaths from pulmonary hemorrhages out of a total of 524 patients treated at the U. S. Public Health Sanatorium at Fort Stanton, New Mexico, with an altitude of 6231 feet. Ten per cent of deaths from pulmonary hemorrhages are not seen in other places.

Sea Climates.—Ancient physicians recommended sea voyages for consumptives. English medical men of the first half of the nineteenth century considered long sea voyages indicated in many cases of tuberculosis. The fact that they have recently been abandoned shows that they have not met with success. But we often meet with patients who want to take a trip around the world as soon as they are told that they are tuberculous. In other cases in which it is desirable to remove

[1] Tr. Nat. Assn. for Study and Prevent. Tuberc., 1908, **4**, 246.

the patient from his home surroundings, the most feasible place is at the sea-coast. In fact, there are many cases in which, as we have just mentioned, high climates are contraindicated, and the patient, anxious for some decided change, asks whether a sea-coast resort is suitable for him. As was already emphasized, we must always consult the preference of the patient and send him to the place he chooses, unless there are strong reasons against it.

It is obvious that the air on the high seas is pure and free from dust and microörganisms; but near the coast it is greatly influenced by the land climate, as well as by the industrial conditions in nearby cities. In fact, in some coast cities it is overloaded with dust and soot owing to mines, mills and factories in the neighborhood.

But its moisture serves the purpose of equalizing the temperature; the seasonal differences are less pronounced. However, to this there are many exceptions, and before selecting a sea-coast resort, it is best to inquire carefully into the local meteorological conditions.

According to Schröder,[1] sea air has a profound influence on the heart and bloodvessels. The cardiac activity is increased and the pulse slowed. He explains it by the action of the strong air currents and the greater heat conductivity of the moist air; despite the decrease in perspiration, the skin is better cooled and the bloodvessels contract. Reflexly, this causes a greater cardiac activity and the peripheral bloodvessels dilate, causing hyperemia of the skin. The result is strong circulation of the blood from the visceral organs to the periphery. The higher air-pressure causes slower but deeper respiration, favoring better metabolism and increased excretion of carbon dioxide. The activity of the skin, and especially of the mucous membranes, is greatly augmented.

Sea voyages are not to be encouraged. "The vicissitudes of sea travel," says Guy Hinsdale, "the narrow cabins, and the difficulty of obtaining a suitable diet, even such common requisites as milk and eggs, should be enough to condemn sea voyages. Tuberculous patients ought not to travel more than is absolutely necessary. Imagine the bacteriological condition of a consumptive's stateroom, for instance, at the end of a month's voyage. What sea captain or steward would ever put such a cabin into sanitary condition for the next passenger?" Then it must be borne in mind that sea sickness is liable to do much harm. I have seen many hopeful cases of tuberculosis take a bad turn after a sea voyage during which they suffered from sea sickness.

As a therapeutic measure sea voyages are therefore to be condemned. But patients who are known to bear the travel well and who do not suffer from sea sickness may be permitted to cross the ocean when necessary. They are, however, to be warned against slow steamers; the sooner they get across, the better; and they must be told that it is best for them to spend the greater part of the time on deck and avoid the close cabin and the stuffy smoking-room.

[1] Brauer, Schröder and Blumenfeld's Handbuch d. Tuberkulose, 1914, 2, 335.

Empirically, it has been found that incipient cases without pronounced constitutional symptoms often do very well at the sea-coast, provided they observe the rules of healthful life. A slight tendency to hemoptysis is no contraindication, but those who show proclivities to copious hemorrhages, especially in the advanced stages, should avoid the sea-coast. Fibroid phthisis, as well as cases of tuberculosis with extensive pulmonary emphysema, are better off at the sea-coast than at the mountains, and I have seen cases relieved or improved, though in inland climates they had been doing badly. Similarly cases with cardiac and renal complications, which cannot be sent to high altitudes, should be sent to the sea-coast when a change is decided upon. Mild implication of the larynx is no contraindication. The cases of asthma and tuberculosis, in which dilatation of the heart is a strong feature, and which are not relieved, or are harmed, at a high altitude, should be sent to the sea-shore where they often recover their strength in a marvellous manner. The same is true of senile consumptives with rigid arteries and rigid chests, in whom paroxysmal attacks of cough and expectoration are occasionally very annoying. They are often benefited by a stay at the sea. Phthisis with chronic bronchitis in which the amount of expectoration is excessive, is relieved at times in a sea climate. Mild forms of neuroses and metabolic disturbances, such as gout, diabetes, obesity, etc., when complicated by tuberculosis, do well at the seashore.

Of course, far advanced cases with hectic or high continuous fever, or with laryngeal, intestinal, and renal complications, as well as acute progressive cases, should not be sent to the sea-coast but should be kept at home.

Desert Climates.—There yet remains to speak of desert climates in which many patients in this country have been cured by "roughing it." These regions may be of low or medium altitude. But their most important characteristic is the capriciousness of meteorological conditions; the changes are quick and extreme. The air is pure—there are usually not enough people to contaminate it—but it is frequently filled with dust and sand, especially after strong winds and storms. Of sunshine there is plenty, often to the detriment of the patient, who finds it hard to contrive a shelter against it.

Because of the frequent changes in the weather, strong, often violent winds, these climates make very great demands upon the reactive powers of the patient, and lead to excessive expenditure of vital force. They are therefore suited only for those endowed with strong constitutions and who have ample recuperative powers. The very young and the very old and those with delicate constitutions should not be sent to the desert. Moreover, patients of the class just mentioned as proper cases for desert climate are not satisfied with climate alone. They demand, as a rule, also social life and amusements to distract them, and these they cannot get in those regions.

It has been found empirically that patients with phthisis compli-

cated by bronchitis and pulmonary emphysema, who expectorate excessively, often do well in these regions. Patients with phthisis complicated by renal disease may also do well, provided there is no arteriosclerosis. Occasionally, we meet a patient in a far advanced stage of the disease who has been "given up," but he decides to discard all comforts and pleasures of life and leaves for some desert region, and within a couple of years returns in excellent condition. These cases are rare, but they do occur. Unfortunately, they admit of no generalization.

A Warning.—Before leaving the subject of climatic treatment of phthisis, I want to emphasize the fact that it is not only good air but also good residence and above all good food that the patient must have if he is to recover. These three in combination are very difficult to obtain. William Garrott Brown, an American historian, who succumbed to phthisis after making a vain fight against the disease, thus describes his experiences:

"It is now seven years and more since I began my quest for a place and an arrangement to breathe freely and constantly the right kind of air, and eat in abundance the right kind of food, yet I can say with perfect honesty that I have not yet found anywhere the combination of these two factors of cure worked out satisfactorily at moderate cost for me and such as I am." He points out that American cookery is peculiarly exasperating—"that is to say, the cooking of such Americans, doubtless the majority, as can be induced to 'take boarders,' and particularly such as can be induced to take boarders who are sick. Many of these last, by the way, are such as have already failed to minister acceptably to boarders who are well. There is, as a rule, not merely unenlightened American cookery; but cookery stimulated by no aspiration and but little competition; cookery seasoned with a lax indifference; cookery without any compelling need to be better, and with an obvious reason for being as careless and unlaborious as it can be and continue to be endured. To take 'lungers' at all, it would seem, confers rather than incurs an obligations. For is not that surrendering the chance of any other kind of gainful hospitality?"

These are the reasons why many patients who have done well at home take a turn for the worse after a sojourn in the country for a few months. Physicians should bear this food problem in mind when sending their patients to boarding houses in the country, and when the place selected has an ideal climate but does not have the facilities for proper housing and feeding the patient, he is safer at home under a carefully regulated open-air treatment, as was already described.

CHAPTER XXXIX.

INSTITUTIONAL TREATMENT.

Sanatoriums.—We have shown that success in the treatment of tuberculosis can only be attained by gaining the confidence and the coöperation of the patient and retaining them over a long period of time, until the termination of the case. The old adage that rest, proper nourishment, and fresh air are effective as curative agents, holds good today for most cases. But they can only be of benefit when taken methodically, and adjusted to the special requirements of each individual case. The tuberculous patient is usually an individual who has not led an exemplary hygienic life, as is proven by the fact that the error of his ways has been instrumental in reducing his natural, and inherent, resisting forces against the ravages of the tubercle bacilli. He must, therefore, be guided into a healthful mode of life. He must also be cared for in such a manner as to preclude the dissemination of the seeds of the disease among those who come into contact with him.

These are some of the reasons why there have recently been established institutions with a view of solving the complex prophylactic, therapeutic, and social problems of tuberculosis. In these "sanatoriums" the patients are under the constant supervision of especially trained physicians who scientifically and methodically guide them along climatic, dietetic, and specific lines of treatment. The rules of rational life are minutely enforced, and the discipline is of a military character in practically all well-conducted institutions.

As soon as a diagnosis has been made, the problem is presented whether the patient should be sent to one of these sanatoriums, or may be cared for at home with an equal outlook for ultimate recovery. In deciding this question it is necessary to take into consideration many factors which are but rarely thought of.

Scope of Sanatoriums.—The first sanatorium was established by George Bodington in 1840, as has already been mentioned (see p. 696). But he failed. Herman Brehmer established the first successful sanatorium in Germany in 1859, at a time when tuberculosis was considered incurable because of the teachings of Laennec, and the experience of ancient physicians. In this country Trudeau established the first sanatorium at Saranac Lake in 1884 and met with considerable success, discharging cured patients, a thing which was in those days considered impossible. With the evolution of our knowledge of the etiology, pathology, and therapy of the disease, the functions of the sanatorium have been greatly enhanced. It has been expected that it

would prove of great prophylactic value by affording places for the segregation and isolation of the bacilli "carriers;" that it would prove of immense therapeutic value because it was assumed that modern methods of climatic, dietetic, and specific treatment can only be carried out under the careful supervision of especially trained physicians; that it would prove of great educational value, teaching the patients a healthful mode of life which is in itself an important weapon in the struggle against the disease, and which may be followed by them after their discharge from the institutions.

With these aims in view, numerous institutions have been established in nearly every country of the civilized world at an outlay of immense sums of money for buildings, equipment, and maintenance. In some countries the State and private insurance companies have provided the funds for the sanatoriums. The fact that within recent years the mortality from tuberculosis has decreased was considered striking proof of the valuable results attained, and the sanatoriums have been given the lion's share of the credit.

But at present, after these institutions have been in existence for over thirty years, we hear inquiries from many competent sources whether they have done all, or the greater part, of what has been expected of them. Articles like that of Edward S. McSweeny,[1] Medical Superintendent of the Sea View Hospital in New York, "Are We Getting Proper Value from Our Plant and Expenditure for the Tuberculous?" are becoming more and more frequent in our medical journals. T. D. Lister[2] is of the opinion that "too much is sometimes claimed as the result of the institutional training of patients." Considering that immense sums of money have been invested in these institutions, it is but proper to inquire whether they have brought returns along therapeutic and prophylactic lines commensurate with the investment.

Limitations of the Usefulness of Sanatoriums.—It seems that the pessimism as to the value of sanatoriums displayed at present is mainly due to the fact that too much was expected from them. They are no panaceas for phthisis. Some enthusiasts, who have advocated their erection and raised funds for the purpose, have in fact promised too much, and when at present these institutions do not come up to the extravagant expectations of some, they are altogether condemned. This is as unjust as the extreme enthusiasm of those who claimed that sanatoriums will solve the tuberculosis problem. In an official report signed by Clifford Allbutt, Lauder Brunton, Arthur Latham, and William Osler,[3] on the value of sanatorium treatment, it is stated: "In many cases, owing to the severity of the disease present, it must be useless; that in a few instances it is actually harmful; and that in many cases this method of treatment need not be carried out in an

[1] Medical Record, 1915, **87**, 94.
[2] Lancet, 1917, **2**, 739.
[3] Ibid., 180.

institution." Bardswell and Thompson's[1] analysis of the experience of the King Edward VII Sanatorium at Midhurst leads to the following conclusion: "The records show that residence in a sanatorium, much though it can accomplish in individual cases, is a means of treatment which is far from being adequate."

Before pointing out the cases in which the sanatoriums may be utilized with benefit in the treatment of phthisis, we shall enumerate some of the shortcomings of this method of treatment:

The number of sanatoriums is inadequate, and we cannot expect that there will ever be a sufficient number to provide for all tuberculous patients, just as we cannot expect that all suffering from active disease can be induced to enter and stay within the institutions until the termination of the affliction. In the available institutions there is hardly place for 5 per cent of the existing proper cases. To provide accommodations for all suitable cases in the United States, several billions would have to be invested in buildings and equipment, and then at least $100,000,000 annually for maintenance. Even the most enthusiastic of those engaged in the campaign for the control of tuberculosis are not hopeful of ever raising such enormous funds.

Sanatoriums are expensive, and it is problematical whether the results attained within them could not be achieved in the vast majority of cases at a lesser expenditure with home treatment. It costs at least $3.00 per day to maintain a patient in a sanatorium. The experiment has never been tried on a large scale to spend that much money on a large group of patients treated in their homes consistently for many months.

It appears that only the very rich or the very poor can afford institutional treatment for months under present conditions. The former can pay any price, and the latter are cared for in enlightened cities by the State, municipal, or philanthropic institutions. But there is a large middle class which will only reluctantly agree to be treated as public charges, as is the case with clerks, small merchants, professional persons, etc., who have been self-supporting until stricken by the disease. They cannot undertake to pay at least $30.00 a week for several months, and at the same time provide for those dependent on them. Neither are they inclined to enter State or municipal sanatoriums, and associate with persons who may be distasteful to them. Only when the disease has advanced far, often beyond repair, and all their own and their friends' resources have been exhausted, do they decide to enter sanatoriums as a last resort, and even then they often leave soon after entering because the surroundings are distasteful to them. This is the main reason why so few incipient cases, derived from these classes, are entering sanatoriums.

It is very difficult to induce patients in the incipient stage of the disease to enter sanatoriums because they maintain that they feel quite

[1] Mortality after Sanatorium Treatment, Med. Research Committee, Special Report Series No. 33, London, 1919.

well and resent the idea that they must live among "sick," or among "consumptives," and they often leave soon after entering for these reasons. *The strict discipline, especially the unavoidable institutional atmosphere, is distasteful to the average human being who will resist all attempts to place him in an institution as long as he can.* The policy of admitting only hopeful cases and discharging bed-ridden or dying patients, does not meet with the success worthy of the effort.

Many patients refuse to enter sanatoriums because they do not want to have the stigma of tuberculosis which, they allege, will stick to them throughout their lives and may interfere with getting employment under present conditions of private and municipal phthisiophobia.

It can be stated without fear of meeting proofs to the contrary that, on the whole, sanatoriums do not show better lasting results than properly conducted home treatment. In this country, hardly any State or municipal sanatoriums have published satisfactory reports with comparative statistics showing the results attained as compared with a similar group of patients treated in their homes. The most competent compilations of statistics have been published by Lawrason Brown and Pope[1] about the discharged patients from Saranac Lake, and by Herbert Maxon King,[2] of the Loomis Sanatorium and by Bardswell and Thompson on the experiences of the King Edward VII Sanatorium at Midhurst. To be sure, Brown shows that five, ten, and even eighteen years after discharge some of the patients were found alive, and even efficient at their occupations. But the average life of the consumptive outside of the institution, under any mode of treatment, has been found to be between six or seven years. Stadler[3] reports that five years after the onset of the disease one-half of tuberculous patients are found able to work without sanatorium treatment. There are similar statistics available for other countries, and I have no doubt that in the United States we would find conditions the same on careful investigation. King's conclusion as to the value of sanatorium treatment is that his inquiry "clearly demonstrates the uncertainty of apparent immediate results of treatment."

This uncertainty refers mostly to relapses, which are to be expected when we consider the undulating course of phthisis, with its periods of remissions and of acute or subacute exacerbations. The few investigations that have been made of patients discharged from sanatoriums in New York show distinctly that a very high proportion have suffered from relapses of the disease, despite the fact that they have been found "apparently cured," or "improved" at the time of their discharge. Many have to be readmitted because of these relapses, and it has been said that the cure is so good and attractive that many patients like to take it several times.

It is clear that patients who die during, or soon after, sanatorium

[1] Am. Med., 1904, **8**, 879; Ztschr. f. Tuberkulose, 1908, **12**, 206.
[2] Tr. Nat. Assn. for Study and Prevent. Tuberc., 1912, **8**, 82.
[3] Deut. Arch. f. klin. Med., 1902, **75**, 412.

treatment are to be deducted from the total number of patients when we want to arrive at an estimate as to the cost of each "cured" case. Dr. Taylor, Tuberculosis Officer, Country Borough of Halifax, reports on "The After Histories of Insured Persons Granted Sanatorium Benefit During 1914, with a Critical Review of the Facts."[1] He remarks that of 1914 applicants treated, 76.5 per cent are dead, and of the remaining, 23.5 per cent half are unable to work. The County of London Committee records the discharge from sanatoriums in 1919 of 1484 ex-service males, of 690 civilian males, and of 761 females. Of the first group 699, of the second, 294, of the third, 293 were unfit for work when discharged; 25, 27, and 23 died in the sanatorium. Midhurst reports that of the 1906–7 discharges 66.4 per cent were known to be dead by 1919. The percentage does not fall below 50 until we reach the 1916–17 discharges, of whom 40.1 per cent were dead by 1919; while of the 184 discharges in 1917–18, 45 (25.4 per cent) were known to be dead, and 21 had been lost sight of. At Newcastle-on-Tyne of those discharged in 1913, 48.4 per cent were known to be dead in 1919, and 18.8 per cent had been lost sight of. Of discharges in 1917, 28.8 per cent were dead and 11.9 per cent had been lost sight of.

In estimating the problem whether sanatoriums bring returns commensurate with the money invested in their erection and maintenance, we must also deduct those cases which suffer relapses, for obvious reasons. And when we do this, in addition to combining with them those who have been discharged because the sanatorium was of no benefit to them, and also those who died, we discover that *the cost per successful case is enormous and hardly attractive to municipal and State authorities.*

The exorbitant cost of sanatoriums is shown in another way. It is well known that from 25 to over 50 per cent of the inmates in the institutions which aim at admitting but "incipient" cases are "closed" cases, with negative sputum. Some authors are inclined to estimate that over 50 per cent of these abacillary cases are in fact non-tuberculous. C. D. Partfit estimates conservatively that 33 per cent of the abacillary cases which are classified as moderately advanced cases of tuberculosis are non-tuberculous. It is on these cases that such large sums are spent with a view of preventing and curing tuberculosis; they improve the statistics of success of the institutions. When we contemplate the cost we are astounded. I assisted at the autopsy on a woman who spent twenty-six years continuously in a sanatorium and a hospital for advanced consumptives. We found that she had no active tuberculous lesion. Even if we count only $500 per year, the community wasted $13,000 on this woman, in addition to the loss of her work which might have been more than this sum, if she had not been kept in an institution. Then, she kept out at least twenty-six patients who really needed hospital care. The sanatoriums and hospitals in

[1] British Med. Jour., 1920, **2**, 216.

this country all have numerous such cases. This is proved by the statistics of Ash and Washburn which we have already quoted (see p. 534).

The educational value of the sanatoriums is beyond question, teaching, as they do, objectively the rules of healthful life. But the patients of the lower social strata, who make up the bulk of dependent consumptives, cannot, as a rule, continue along the hygienic lines which they have learned. Returning to the tenements, with rooms without windows or baths, coupled with a low earning capacity, one cannot live in the manner he learned in an institution. Relapses, which are likely under all circumstances, are inevitable for these reasons alone. In England Dr. Lister and many others have considered the educational value of sanatoriums a great failure.

On the other hand, the aggressive campaign, recently carried on by the various antituberculosis agencies has done all that can be done along educational lines. In fact, the dispensaries with their social services, the day and night camps, etc., achieve educational, as well as therapeutic results which are, from a certain viewpoint, superior to, and more far reaching, than those of the sanatoriums, and at less cost.

Let us not overestimate the prophylactic value of the sanatoriums. It was hoped that by segregating consumptives, sources of infection would be isolated. But we have already shown that this was a vain hope. Only "incipient" cases are admitted—so far as they can be found and induced to enter in time—while advanced cases, which are the most dangerous, because they expectorate myriads of tubercle bacilli, are rejected. The statement that institutional treatment is the predominant cause of the decline in the death-rates from phthisis, which has been expounded by Newsholme[1] with such vigor, is not supported by facts. Newsholme's figures have been demolished by Karl Pearson,[2] one of the most competent authorities to judge statistics. In Germany—the home of the sanatorium—this claim has been abandoned during recent years. As was pointed out by Cornet and Robert Koch at the Antituberculosis Congress in London, there were at least 226,000 persons disseminating tubercle bacilli in Germany, and only 20,000 were cared for in institutions, and of these latter only 4000 expectorated bacilli. This number could not have had any perceptible influence on the morbidity and mortality from tuberculosis. In the United States conditions are the same. In recent attempts at prophylaxis of transmissible diseases no attempts are made to isolate cases when the number of "carriers" is large. This point has been very well elaborated by one of the best sanitarians, Charles V. Chapin. Why tuberculosis is an exception has not been shown. Indeed, it has been aptly stated that it would be much easier to segregate those who

[1] Prevention of Tuberculosis, London, 1908.

[2] Fight against Tuberculosis and the Death Rate from Phthisis, London, 1911; Tuberculosis, Heredity, and Environment, London, 1912.

had not been infected with tubercle bacilli than those who harbor this virus in their bodies. The number of the former is much less. (See Chapter III.)

From the clinical standpoint, we are not in possession of reliable statistics showing that the mortality of patients who have been treated in sanatoriums is lower than that of those who have been cared for in their homes. The statistics of experiences in English sanatoriums, quoted above, seem to show that it is as high as could be expected if no treatment had been instituted. We have already mentioned that the institutions in the United States have not published comprehensive data along these lines, excepting those of Lawrason Brown and King. In Germany, although long and apparently learned books and articles have been produced, they are just as much in the dark about this problem as we are in this country. The reasons are that the material is not comparable. A drastic illustration may be cited. In the selection of cases it is aimed at admitting only those in the incipient stage. The result is that at Grabowsee 45.2 per cent, and at Melsungen 97 per cent of the patients have not shown any tubercle bacilli in the sputum. Ulrici reports that in 40 per cent of the patients at Mulrose he could not make a positive diagnosis of tuberculosis, and Leube says that many patients who are admitted to sanatoriums in Germany are, when examined by military surgeons, found fit for the army and accepted. And during the World War, the military authorities found that a large proportion of these consumptives made excellent soldiers. To be sure, the outdoor life and the regularity in habits which military service involves, as well as the nourishing food, may be some of the factors in improving many tuberculous patients, as some have suggested. But it seems to me that the greater number of these patients, though they had been in sanatoriums, were not at all tuberculous. They are derived from the class collectively grouped as "consumptives with negative sputum.

It is obvious that statistics of such "consumptives" will show good and lasting results of treatment. In their book on the prognosis of tuberculosis Kuthy and Wolff-Eisner, reviewing the subject, say that exact and scientific data are not available to prove the value of sanatorium treatment; and Newsholme, who is a great believer in the benefits of institutional treatment, also says that there are no exact and comparable data available to prove it.

Causes of Failure of Institutional Treatment from the Therapeutic Viewpoint.—While institutional treatment undoubtedly has its advantages, which will be shown later on, it is by no means the best, and clinicians cannot approve of all the methods pursued in sanatoriums. The fact is, *wholesale treatment of such a complex disease as phthisis is not ideal. Individualization is here of greater importance than in most other diseases.* Says Albert Robin:[1] "One of the disadvantages of

[1] Traitement de la tuberculose, Paris, 1912, p. 67.

the sanatorium is that it applies too often arbitrary principles to patients whose disease can only be relieved by individualized methods. It is for this reason that the practitioner who knows how to adapt the treatment to each of the small number of patients under his care, and to take cognizance of the temperamental indications, is qualified to manage a case of tuberculosis as well as, if not better than, the sanatorium doctor who has under his care a large number of patients of whose individual idiosyncrasies he is ignorant, at least for a time, and must therefore have a strong tendency to subject them all to the same method of treatment." This refers to private sanatoriums, in which the patients must be catered to if they are to be retained for months. In State and municipal sanatoriums, where the poor and dependent patient faces starvation if he leaves the institution, the trouble is of a diametrically opposite character. The fact that a large proportion of patients leave before the physicians consider them fit for discharge, shows that they cannot be satisfied.

This lack of individualization in treatment is seen in many ways in the sanatoriums which are hotbeds of therapeutic hobbies. But this is usually not so harmful as the uniformity of the diet in institutions. *Mass feeding is difficult at best and can only be carried out in jails, where the inmates have no choice, or in armies during war.* In a discussion on the sanatorium problem in England, T. D. Lister[1] thus summarized the food question: "It is badly cooked, badly served, from ignorance or lack of sympathy for human weaknesses, from unnecessary monotony in the daily menu. For the convenience of the staff and store room there is a melancholy recurrence of the same food after the same intervals in some sanatoriums. Loss of interest and loss of appetite result. Patients, staff, and doctors often become institutionalized. There is always a risk of deterioration in official clinicians." To subject to the same dietary tuberculous patients in different stages of the disease, with different individual capacities for digestion and assimilation, who have been brought up on, and adapted to different kinds and preparations of foods, is bound to meet with failure. For this reason we find that complaints about the quantity and quality of the food are universal in public sanatoriums, and to some extent in private institutions where food is served *à la carte*.

It can hardly be expected that municipal, State, and philanthropic sanatoriums should supply food *à la carte*; it will always be *table d'hôte*. And for this reason resentment on the part of the patients is to be expected. To be sure, these institutions are always filled and there are long waiting lists, though during recent years the latter seem not to be such a striking fact as it was formerly. But when patients leave before they are discharged, we may safely assume that the cost incurred during several weeks or months for their maintenance was, to a large extent, wasted.

[1] Lancet, 1917, 2, 739.

In American municipal sanatoriums of the large industrial cities the failure in this regard is even greater than in other countries, because we must care for tuberculous immigrants of various nationalities, whose tastes differ extremely as regards food and its preparation, as is shown elsewhere in this book.

These are some of the drawbacks of sanatorium treatment. It is for these reasons that the municipal and State sanatoriums in many cities of the United States are not filled with a desirable element, but contain a large proportion of undeserving individuals. "My efforts are not going to be devoted to coddling tramps and other parasites," exclaims in despair Dr. Edward S. McSweeny, the Medical Superintendent of the Sea View Hospital in New York. These are also the reasons why the best elements of the tuberculous population in this country will always have to be cared for in their homes, as is the case at present.

Indications for Institutional Treatment.—But there are many cases of tuberculosis which cannot be treated in any other place than in institutions. In fact, anyone with experience in a large city is convinced that tuberculosis cannot be managed without the aid of institutional treatment. Of the cases which are suitable for sanatorium treatment and would be lost without it, we may mention the following:

Among well-to-do patients we meet with many who, for various reasons, cannot be cared for in their homes. To send them to the country without control may prove disastrous, because the foolish and reckless rich show at times greater lack of self-restraint than the stupid poor. They are best cared for in private sanatoriums in which most of the drawbacks of the public institutions are eliminated. They may be sent to sanatoriums for a short stay, over the hot summer months, or for outdoor treatment for the relief of an acute exacerbation, etc.; or for a long period till the disease is arrested. Great care should be taken that they do not become egocentric, excessively introspective, or hypochrondriacs, which is not unusual.

Among the poor, and those who have become dependent because of the disease, we meet with a large number of patients who have no family to care for them during their illness and, with or without funds, they are unable to find lodgings under present conditions of rampant phthisiophobia. Many boarding houses bar persons who cough; and at times even near relatives are overtaken with a sense of stupid fear of infection, and want to get rid of the unfortunate patient. For these there is left nothing but to go to a well-regulated sanatorium.

There is a large number of phthisical patients who notoriously lack will power to carry out the most important of the measures prescribed for them and, remaining in the city, they are apt to be tempted by the opportunities for gay life, or even excesses. They are better off in sanatoriums.

On the other hand, there are many who show all willingness to do everything that is conducive to the cure of the disease, but they

have not the funds to pay for capacious rooms in a desirable part of the city, for good nourishment and medical attendance. Tuberculosis is after all the most expensive of diseases, not only for the special and costly nourishment and residence which are required, but mainly for the long time the patient must remain idle, and the savings of years may be exhausted before he can resume work. While most of these can be, and are, well cared for in the clinics, the day and night camps, found in every large city at present, we meet with many who, for obvious reasons, are better off in sanatoriums, at least for short stays.

Most phthisical patients should leave the city during the hot summer months, and those who cannot raise the funds for the purpose are proper charges of the sanatoriums. Indeed, if the sanatoriums were not filled with lazy, undeserving tramps and vagrants who remain for years in the institutions, and when discharged from one, soon gain admission to another, they could well care for the just mentioned class of patients. It seems to me that the German system of admitting tuberculous patients for three or four months is much superior to ours, where they are often kept indefinitely. The result is that the patients must wait for months before beds are vacant for them, and truly incipient cases, left without proper care while waiting for admission may become advanced.

The longer we are up against the problems presented by tuberculosis in the city, the more we are convinced that *the public sanatoriums ought to be converted into hospitals which admit patients on short notice, keep them for a few weeks, a month or two, until they regain their strength, and are fit for treatment in the clinics.* Patients who suffer from acute exacerbations during the long, chronic course of phthisis could then be cared for. Inasmuch as municipal institutions are now in abundance near cities, this could easily be accomplished.

But sanatoriums still work on the theory that they are to cure their patients, which they cannot do in more than 5 or 10 per cent of cases, which is, in fact, not more than home treatment accomplishes.

CHAPTER XL.

DIETETIC TREATMENT.

Economic Aspects of Dietetics for Consumptives.—Because phthisis is accompanied by wasting of the body it requires careful, generous, and at times excessive nourishment with a view to covering the deficit created by the extravagant drain resulting from the toxemia, fever, loss of appetite, disturbed digestion, faulty metabolism and concomitant emaciation. Cornet suggests that the rapid waste of the tissues tends to hasten absorption of the proteins surrounding the tuberculous foci and thus, at the same time, inhibits the natural process of healing by means of induration and also furthers the peripheral dissemination of the bacilli. Inasmuch as the disease finds most of its victims among the poor and destitute, or causes destitution and despondency in those who had been self-supporting before its onset, the dietetic problems are not only of a physiological nature, but also have important economic bearings. It is self-evident that a dependent consumptive must not be prescribed food which is beyond his reach financially.

In my experience the dietetics of phthisis is, in fact, more dependent on the financial resources of the patient than on the careful calculation of the number of calories contained in the various foodstuffs. Considering the variety of dietaries which have been urged in this disease by various authors, and that each author claims good results with his method, it is obvious that *no specific diet has been devised which will suit every case. In fact, all that can be stated is that tuberculous patients need food, just like other persons who are underfed, but they usually need more of it.*

Need for Individualization of Diet.—Most of the studies in the dietetics of phthisis have been carried out in sanatoriums, some of which have had sufficient funds for an extravagant diet, while others with meager finances are alleged to have shown similar results. But the lessons from institutional experience are not applicable in their entirety to patients treated in their homes. On the other hand, the time-honored advice given to tuberculous patients: "Eat plenty of milk, eggs and meat," is often decidedly harmful to those who follow it implicitly.

There is great urgency for individualization of the diet in phthisis; it is important that the diet should be adapted to the needs of the patient, and not to the disease. The "personal equation" counts for more than the disease.

There is no doubt that the failure of institutional treatment of

phthisis is, in a large measure, due to negligence in this regard. Wholesale feeding is usually disastrous for human beings. The food in first class *table d'hôte* restaurants is usually unbearable to the average healthy person, when relied on continually for a considerable time. It is impossible to make up a menu which will suit the palate, digestive capacities, and functions of one hundred patients in an institution where they must remain for months. The difficulties are greater with tuberculous patients whose gastric function and capacity for assimilation of ingested food are very often deranged. Tuberculous patients cannot be treated like soldiers in the army, or prisoners, if we are to succeed in our aims.

It is not true that two kinds of food of different composition, but theoretically of the same nutritive value, will invariably be of the same digestibility, or produce the same effects as regards nutrition. It may be calculated in the laboratory that a portion of beefsteak, roast beef, poultry, sausages, stew, cheese, potatoes, cereals, bread, milk, eggs, etc., contains a certain proportion of proteins, fat, and carbohydrates, and will liberate a certain number of calories when burned in the body. In fact, we know that the intrinsic value of three eggs is equivalent to about 100 grams of red meat, while 100 grams of bread is approximately equal to one egg, or 30 grams of beef, or 200 grams of potatoes, or 280 grams of milk. But very often a consumptive assimilates three boiled eggs more easily than 100 grams of beef, or 300 grams of bread. At times the patient assimilates 250 grams of milk better than 200 grams of potatoes. On the other hand if he has tuberculous ulcerations of the intestine he may not be able to digest and assimilate milk and eggs, but beef or poultry are digestible. Because of the personal equation many patients refuse to thrive on scientifically prepared dietaries. An Irishman resents spaghetti, an Italian refuses Irish stew, a German prefers sausages to the English roast beef, etc.

For these reasons, in prescribing a diet for a patient we must always take into careful consideration his habits of life, the foods upon which he has been raised, and his personal likes and dislikes. Even when a change is imperative, it is dangerous to institute it suddenly, and we must make a strong effort to fit the diet to the one the patient has been used to. The factors which should guide us are the presence or absence of anorexia, fever, constipation, diarrhea, etc.

Superalimentation and Forced Feeding.—With a view of replenishing the wasted tissues, especially in those who are by nature bad eaters, it has been suggested that superalimentation, or even forced feeding, is indicated in most cases of phthisis. It has been observed that occasionally an emaciated patient gains in weight under such a regimen, and some authors have advised that all sufferers from phthisis should be "stuffed." Even Debove's method of introducing food through the stomach tube into those who would otherwise not consume large quantities of nourishment was in vogue for some time until it was

found that the gain in weight which forced feeding produced in some cases was not necessarily an indication that the lesion in the lung had improved. It was also found that many patients under forced feeding, with or without the stomach tube, may gain in weight and improve otherwise for some time, when suddenly the gastro-intestinal tract rebels, and within a few days they lose more than they had gained in several months.

Estimation of the Nutrition of the Patient.—In our attempts at estimating the results of certain dietetic methods in tuberculosis we cannot always be guided by the scientific determination of the number of calories ingested by the patient every day; nor even by the quantity of proteins, fat, and carbohydrates which the patient has consumed. Attempts along these lines have proved futile in practice; they have not given us a diet which will suit all, or the vast majority, of cases. It seems that only clinical observation of the individual patient, his state of nutrition, his digestive capacity and the assimilability of the ingested food are of value in this regard.

We aim at increasing the amount of nourishment so that the patient shall gain in weight and remain stationary at somewhat above his usual, or normal, weight before the onset of the disease. While in the vast majority of cases a gain in weight is a good index of the value of the diet, it is, however, often liable to mislead. *Fattening by no means goes hand in hand with enhancing the resistance against the tuberculous toxemia in every case.* We meet with cases with hardly any gain in weight, in fact remaining under the standard weight, yet the lesion in the lung heals, and recovery is good.

"The main object of dietetic treatment," says Brown,[1] "is to enable the patient to regain his lost weight, but not to make him a flabby, breathless mass of inert fat." Excessive nourishment, which increases the weight of a patient more than two or three pounds per month on the average, is apt to result in an overload of fat and water without any utility. We should strengthen, but not fatten the patient. "When a workman has to perform hard work, he eats meat," says Daremberg;[2] "the consumptive has to perform a very hard task, the task of repairing his wasted body." In fibroid phthisis obesity is not rare—"obésité toxique" of the French—and is often more annoying to the patient than the symptoms in the respiratory organs.

In the average case we may judge the progress of the disease by following the weight of the patient, provided we also take other factors into consideration. With the increase in weight there should also be an increase in strength; physical examination should show regression of the signs in the lungs, the cough should be ameliorated, and the quantity of sputum decreased. With such signs, a slow and persistent gain, finally reaching ten to fifteen pounds higher than the patient's

[1] Osler's Modern Medicine, 1, 482.

[2] Les differentes formes cliniques de la tuberculose pulmonaire, Paris, 1905, p. 149.

normal weight before he was attacked by phthisis, indicates that we may be satisfied that the diet is good.

Do All Tuberculous Patients Need Special Diets?—A large proportion of phthisical patients, probably one-third of all, have good appetites and digestion. In fact, even febrile consumptives are seen without anorexia which accompanies nearly all other fevers. The prognosis is good so long as they retain their normal gastro-intestinal functions. They may be told that a moderate increase in the quantity of food they have been accustomed to eat is sufficient and, when possible, they should increase somewhat the quantity of proteins and fats, provided the stomach does not rebel.

If the constitutional symptoms are in abeyance, or disappearing, and the signs in the lung show that the lesion is cicatrizing, we should not worry about a lack of gain in weight, or even when they remain a few pounds less than their normal weight. *A patient with a good appetite and digestion needs no special diet;* he should eat just like any other person, or a little more, if he can without inconvenience. On this point all authorities agree today. Thus, King[1] says: "In the absence of certain complications, a diet which would suffice for the same individual under normal conditions of life will doubtless, with very slight modifications, meet the requirements in the presence of tuberculosis, the more especially during that period of the disease when constitutional symptoms are either absent, or but slightly manifest." Paterson,[2] whose patients work at graduated labor, gives them "a liberal diet which consists of the ordinary food which the working classes provide for themselves when they are in a position to afford it." In fact, patients who tend to become excessively fat have their diet reduced in quantity.

On the other hand, patients who lose progressively in weight and strength, are anemic and debilitated, despite the rest which is rigidly enforced, need more and better food if they are to recover, or hold their own, in the struggle with the disease. But even here superalimentation must be carefully adapted to the digestive capacity of the patient.

It may be stated, as a general rule, that the suggestion of some authors that in such cases the patients must consume between 4500 and 6000 calories daily is a dangerous one. Experience has taught that one who will not recover, or hold his own, on a diet of 3500 calories, will not recover at all. N. D. Bardswell and John E. Chapman[3] have arrived at the conclusion after a thorough experimental study of the subject that 3000 calories per day are quite sufficient.

Variety.—The first principle to be observed in the diet of the tuberculous patient who is losing weight is variety, both as regards nutritive principles as well as appetizing qualities. There is nothing more abhorrent to a tuberculous patient, and to a large extent to all sufferers

[1] Sixth Internal. Cong. Tubere., 1908, **1**, 719.
[2] Ibid., p. 893.
[3] Diets in Tuberculosis, London, 1908.

from chronic diseases, than homogeneity of diet. No limited and exclusive diet can keep a patient well for any length of time because it does not respond to the urgent demands of the different organs and tissues of the body. It does not stimulate the secretions of all the digestive glands. If an exclusively animal diet is taken, only the gastric juice is stimulated, while the saliva, pancreatic juice, bile, and intestinal juices are not utilized and, remaining free in the gastro-intestinal tract, are apt to act as irritants and produce diarrhea which is exhausting, or constipation which is harmful in other ways.

We often meet with patients who have been given diet lists in which four or five meals are listed for the day. But any appetite they may have had before the list was consulted promptly disappears, because it shows the foods which have been given them for months without any appreciable variation. Many patients who have followed the injunction "plenty of milk and eggs" have engendered such an aversion to these articles that the mere mention of an egg is sufficient to disturb the slight appetite for other foods which was called forth by hunger. It is always advisable to consult the patient as to the kind of food he prefers or longs for and, if there are no contraindications, to give it to him.

Precautions to be Taken when Overfeeding Patients.—Before a patient is urged on to a course of superalimentation certain precautions are to be taken: He must be carefully examined with a view of ascertaining whether or not he can stand additional feeding. Those showing signs of arteriosclerosis, nephritis, gall-stones, nephrolithiasis, or gout, should not be allowed superfeeding, especially with animal proteins. It is likely to throw a considerable strain on the kidneys, or even produce albuminuria. Moreover, as we have seen, patients with these diseases do rather well when affected with tuberculosis (see p. 602). The condition of the stomach is to be ascertained, and those having dilated organs, or disturbances in the tonicity and motility of the viscus, are to be treated for these troubles when practicable. The appetite is of great importance. Although we may succeed with some patients in urging them to eat irrespective of the appetite, we will fail with many.

Proper preparation of food goes a long way in counteracting anorexia; Dettweiler, who made a great success with his sanatorium, said that the kitchen was his pharmacy. It is to be regretted that very few, if any, modern public sanatoriums are conducted on this principle. It is better to give the patient small quantities of each of several dishes, well, and appetizingly prepared, than large quantities of one or two dishes. The fact that the food value is theoretically sufficient in the latter case does not alter matters. With some patients animal food should predominate, with others eggs, and with still others, milk. The diet must be frequently changed, especially when the digestive tract shows signs of rebellion.

With well-to-do patients these are simple matters, but with those

of limited means this problem is often hard to solve. The writer usually sends for the mother, wife, or sister of the patient and gives her directions along these lines.

Bearing in mind that the disease is likely to last for months, if not for years, we must spare the digestive organs, the cornerstone of phthisiotherapy, as they have been called, and not overburden them with work. The first imperative principle is proper mastication. But regularity in meals is of the same importance. The menus of some authors mention six and more meals a day, which are excessive, in my experience. Three, at most four, meals a day are sufficient for most patients, and afford some rest to the stomach between the meals. At all events, the stomach must be given a complete rest during the night, which can be done by avoiding all food between 9 P.M. and 7 A.M.

Protein Foods.—Experimental researches of Richet and Hericourt, and others, have proved conclusively that when ingested raw, animal foods have an especially beneficial effect in tuberculosis. The specific effect seems to reside more in the juices of the meats than in the fiber. Herbivorous animals, like the cow, are more prone to tuberculosis than carnivorous animals, as the dog.

The best source of proteins for a tuberculous patient is animal food; the proteins of vegetable origin are not so easily assimilated. Meats possess all the qualities which are necessary for the nutrition of the consumptive. To be sure, there are some who maintain with Kellogg[1] that a low protein diet is productive of better results, and urge vegetable proteins in the dietetic management of the malady. It is, however, an every-day observation that the animal proteins do not tax the digestive organs to excess and, excepting in those who suffer from some form of dyspepsia, they can be taken by most consumptives without difficulty in comparatively large quantities. Beef, mutton, lamb, poultry, game, fish, oysters, eggs, milk, cheese, etc., offer a wide range of choice for variety.

Those who have no natural abhorrence for raw meat may have it with great benefit—*zomotherapy* was at one time very popular, especially in France, and may be utilized, when tolerated. Some patients are not averse to taking a small piece of raw beef, dipping it in tomato sauce, and eating it. It is, however, better to mince or chop it, and eat it between two slices of bread as a sandwich, but it should be seasoned to taste. The vast majority of patients, however, prefer roasted or boiled beef, mutton, poultry, etc. It must be mentioned that when roasted or broiled, meats should be rather underdone and, on the whole, they should be changed often in kind and in the form prepared for the table.

But it should never be excessive; we cannot rely on animal foods exclusively in nourishing a tuberculous patient. To supply a patient with 5000 calories per day, it would be necessary to gorge him with

[1] Sixth Internat. Cong. Tuberc., 1908, **3**, 740.

six and a half pounds of meat, or thirty-six eggs, or five quarts of milk, or two pounds of cheese. This would be too much—no human being could take it with impunity for any length of time. For this reason other foodstuffs are necessary in addition to the animal food. The most the average consumptive should have is about three-fourths to one pound of animal food, and when meat is taken raw, it should not exceed one-half pound per day. When this is taken with one pound of bread, three eggs, one quart of milk, eight ounces of potatoes, and four ounces of fresh vegetables, and some fruit, the diet is complete.

Attempting to feed tuberculous patients with proteins we are often confronted with the high cost of animal foods. In many cases we may attempt to supply proteins from fish, which are much cheaper than beef, veal, or poultry. Now, beef contains from two to three ounces of protein per pound. Fresh fish, such as haddock, cod, halibut, perch, salmon, mackerel, or shad, contain from one-and-a-half to two-and-a-half ounces, while the commoner dried fish contain even more proteins, up to three ounces per pound. Proteins in a digestible form may thus be purchased, when fish are used, at from 30 to 50 per cent of the cost of the same amount when consumed in meats. In fact, Drummond[1] has shown that the coagulable proteins of the muscle tissue of cod, herring, and canned salmon have a nutritive value as high as those derived from beef. The empiric experience of the therapeutic value of cod-liver oil in tuberculosis has also recently been reinforced by the investigations of Osborne and Mendel[2] who discovered in fish one of the important vitamins, the fat-soluble A, which has such an immense influence on the growth and health of the animal body. Drummond urges, therefore, the so-called "fatty" fish, which contain considerable quantities of fat distributed throughout their muscle tissue, as important foods containing the fat-soluble vitamin. The herring is especially to be recommended from this angle. Either for the sake of variety, or because of imperative saving in cost, fish should not be neglected from the diet of the tuberculous.

A consumptive needs more protein foods than a healthy person because the disease destroys the tissues, especially the muscles, and there are no better tissue builders than proteins. But we must not give them at the expense of other foods. It is unnecessary, even dangerous, to give more proteins than are required for repairing the tissues; otherwise they are likely to prove more disastrous than to a healthy individual. These evils are, as the researches of Chittenden, Mendel, Folin, Herter, Metchnikoff, Tissier, Combe, Turk, and others show: (1) That protein which is not used for tissue building is not "burned clean," as are fat and carbohydrates, which yield merely water and carbon dioxide, but leaves behind "clinkers" in solid form—for instance, uric acid; (2) that meat proteins also contain such "clinkers" in their extractives, which are superadded to the similar products from the

[1] Jour. of Physiology, 1918, **52**, 95.
[2] Jour. Biol. Chemistry, 1914, **17**, 401.

metabolism of proteins in the body; (3) that all protein which is not absorbed is subject to putrefaction in the intestinal canal, and gives rise to toxins which are partially absorbed and produce injuries of various kinds to the organism (Irving Fisher).

Milk.—Milk has been considered for centuries a good food for consumptives—Aretaeus, eighteen hundred years ago already spoke of it favorably in this connection. It contains more than 10 per cent of nutritive matter, albumin, fat, sugar, and salts. But this does not mean that it is good to use it exclusively for our patients as has been done in the well-known "milk cures." If we wanted to supply all the requirements of a patient it would be necessary to make him ingest five to seven quarts of milk per day. In a few weeks his stomach would be dilated two or three times its normal dimensions.

But with other foodstuffs it is excellent because its nutritive principles are easily digestible in the stomach and intestines, and it contains no toxic substances. It is just as good for a patient with fever as for one who is afebrile. A quart of milk is equivalent in fuel value to a pound of lean meat, or eight eggs. It is thus evident that, from a certain standpoint, it is a much cheaper source of fuel than either meat or eggs.

It is best given between meals in the form of drink, and may be added to many other foods, especially cereals. But it must not be abused; patients who gorge themselves excessively with milk lose their appetite for other foods. Between a pint and a quart of milk per day is to be considered the maximum for the average patient.

There are patients who do not bear milk very well. In some it provokes lactic and butyric acid fermentation in the stomach; this viscus becomes dilated, and the complicating hyperchlorhydria favors spasmodic contraction of the pylorus. In others, the milk clots excessively in the stomach, large solid curds are formed which irritate the mucous membrane and cause nausea and vomiting. In some patients the milk passes the stomach without difficulty, but it produces trouble in the intestines—gaseous distention and diarrhea. I have seen many cases of diarrhea in consumptives, which were thought to have been caused by intestinal ulcerations, but which disappeared with the withdrawal of milk from the diet.

The milk may be rendered more digestible by diluting it with alkaline waters, or lime water, but then the total quantity consumed must be reduced. It is usually more easily digested when given with some cereal, like oatmeal, or rice. Atwater found that milk is more easily digested when it is part of a mixed diet. When consumed alone the proportion digested was: proteins, 91.2 per cent; carbohydrates, 86.3 per cent; and fat, 92.8 per cent. When milk and bread made up the diet, the amount digested was: proteins, 97.1 per cent; carbohydrates, 98.7 per cent; and fat, 95 per cent.

Fermented milk is often more easily borne in large quantities when the pure article is not sustained. We may try koumiss, keffir, or the

various preparations of buttermilk, which are at present supplied by most milk dealers at reasonable prices, or may be prepared at home with cultures or tablets of lactic acid bacilli.

Cheese is an excellent food for consumptives. But we should avoid the highly seasoned varieties. Cream cheese and ordinary pot cheese contain considerable nutritive elements and do not provoke cough or gastric irritation.

Eggs.—Eggs are considered an excellent food for tuberculous patients by the profession and the laity. In assimilability they exceed any known food excepting milk and oysters. They contain enormous quantities of albumin and fat. The white of an egg consists of pure protein which is as digestible and nourishing as that of beef; the yolk contains 25 per cent of fat, 15 per cent of protein, and also nuclein, lecithin, iron, and salts. Eating one dozen eggs per day, a consumptive could feed himself, and pushing it to twenty eggs he would absorb the equivalent of two and a half pounds of beef, because an egg of 50 grams is equivalent to about 35 grams of moderately fat beef, or 128 grams of cow's milk. In other words, they contain over 700 calories per pound; the whites yield 250 and the yolks 1700 calories per pound. But an exclusive egg diet is just as bad as an exclusive meat diet. Too much fat is introduced into the stomach and congestion of the liver is the result, while with an exclusive meat diet, congestion of the kidneys occurs.

It appears, however, that eggs have been abused as an article of food for the tuberculous. Many of the gastric derangements of tuberculous patients can be traced to the abuse of eggs as a food. Most patients consume them raw, and it has been found that raw white of egg is decidedly indigestible. Mendel and Lewis[1] pointed out that, when given to animals, raw eggs give rise to diarrhea. W. G. Bateman[2] found that in dogs, when given in considerable quantities, it sometimes causes vomiting and invariably produces diarrhea. Pawlow found that raw white of egg only partly stimulates a flow of gastric juice. But Bateman shows that cooked egg-white, on the contrary, calls forth an abundance of juice and unites easily with hydrochloric acid. Egg-white remains but a while in the stomach, and escapes in gushes through the pylorus. "Once in the intestines the native egg-white continues to oppose the digestive enzymes, for it has remarkably strong antitryptic properties. . . . Not only does it resist digestion itself, but it prevents the digestion of other easily digested proteins." It is very poorly utilized. In large doses, from 30 to 50 per cent of that ingested is wasted by being ejected with the feces. In normal feces albumin is never found. In contrast with egg-white, egg-yolk has been found to be well digested and utilized.

Clinicians who have the care of tuberculous patients should therefore heed the following warning of Bateman: "A substance which

[1] Jour. Biol. Chem., 1913, **16**, 55.
[2] Ibid., 1916, **26**, 263; Am. Jour. Med. Sci., 1917, **153**, 841.

fails to stimulate a flow of gastric juice and is antipeptic, which hurries from the stomach, calls forth no flow of bile, and strongly resists the action of trypsin, which is poorly utilized and may cause diarrhea, has evidently little to recommend it as a foodstuff of preference for the sound person, let alone for the invalid."

On the other hand, cooked egg-white is easily digested and well utilized by the economy. All that is necessary to prepare egg-white for digestion is to heat it to 70° C. Under no circumstances should a tuberculous patient be permitted to consume several raw eggs a day. They should invariably be boiled. In fact, in my experience eggs may be fried, scrambled, or prepared in any way; so long as they are not consumed raw, they make an excellent food for tuberculous patients.

But there are some exceptions. Those who suffer from derangement of the function of the stomach and the liver do not bear eggs very well and they may have to be discarded. The same is true of patients who have an idiosyncrasy to eggs and get colicky pains in the abdomen, vomiting or diarrhea from an egg.

Four to six eggs per day is about the maximum which a patient should be allowed, if we are to retain the functions of the stomach and liver. In most cases less should be given.

Fats.—While the amount of fat necessary for the average consumptive has been exaggerated by many authors, it is nevertheless a fact that a diet containing a surplus of easily assimilated fat is the best. It must, however, be borne in mind that the capacity for digesting and assimilating fat varies with the individual. In some patients an increase in the amount of fat is immediately followed by gastro-intestinal disturbances. Many people cannot digest fat meats like bacon, ham, etc. We have already mentioned that many patients have shown intolerance for fat even before the onset of the disease.

I have found that butter is superior for our purposes, and it has given me results as good as cod-liver oil, which has been popular for centuries. I direct my patients to cut their bread in thin slices and cover them with heavy layers of butter; mixing butter with mashed potatoes and other foods. As much as six to eight ounces of butter can thus be consumed daily by the average patient without gastric or intestinal disturbances. Those who like to and can consume large quantities of unskimmed milk may get the greater part of their fat in this manner, while cream and certain kinds of cheese are also rich in fat. In looking for sources of easily digestible fat we must not forget fish: salmon, pompano, sardines, shad, fish roe, caviar, etc., are very good for this purpose. Those who have great tolerance for fat may also take in addition to butter, cream, cream cheese, fat meat, and bacon.

The quantity of fat a patient should consume varies according to the season, the kind of food he has been accustomed to eat, h s tolerance of fat, and the condition of his gastro-intestinal tract. Of course, those who are obese, and they are not rare among those with quiescent and healed lesions, should be discouraged from eating an excessively fat diet.

It has been my experience that a patient without preëxisting gastric disease can consume six ounces of fat every day for months with benefit. But now and then one is met who shows a decided inclination to fat intolerance. It is my impression that in most cases it is due to the excessive amounts of improper fats which have been forced upon them. It has been suggested by Tibbles that when a patient cannot take fat, the proteins can be increased; 100 grams of proteins will yield 40 grams of fat. Proteins alone will never fatten a patient; 6.5 pounds of lean meat, or 5.5 pounds of lean and fat meat would be required to supply the daily requirements of carbon for an ordinary person; therefore some other source for fat must be found.

We must guard against quick fattening, "stuffing," of tuberculous patients. Often consumptives are urged to eat plenty and some ingest enormous quantities of food and gain remarkably well. Taking their weight weekly, and finding that it keeps on increasing, they are encouraged to continue in this manner, and at the end of three or four months the gain may be as much as thirty or even forty pounds. But to their dismay they have not been rehabilitated in other respects; they are as yet unable to work, and are, in fact, weaker than before. The weight they have put on is only an added burden, which is not only useless, but actually incapacitating. In addition, they suffer from annoying dyspnea. Physical examination shows that the process in the lungs has not improved; perhaps it has distinctly extended. Carefully and guardedly reducing these patients has often been of great benefit.

Carbohydrates.—In the eagerness to supply the body of the patient with proteins and fat, carbohydrates must not be neglected from the diet. They are, as a rule, easily digested and assimilated, and they spare the proteins, thus maintaining the nitrogen balance, or equilibrium, with smaller quantities of proteins. The best sources of carbohydrates are potatoes, cereals—like oatmeal, rice, etc., which may be taken with milk or cream—pastries, and above all, bread. Cane sugar and maple sugar, which enter into various culinary preparations, are of great value. Daremberg,[1] however, objects to excessive consumption of sweets by consumptives because they are usually dyspeptics who do not stand it very well. He says that those who can take an excessive quantity of sugar may become fat rapidly; but this fattening is not lasting, just as the fattening obtained from an excessive milk diet. The best fattening is obtained from a mixed diet. However, there is no reason against eating sweet desserts, or even candies, in moderate quantities, provided they are taken after meals when they are not likely to interfere with the appetite.

Salts.—Mineral salts must not be neglected. Even if the theory of demineralization is not well founded, there is no question that the loss of mineral salts is higher in consumptives than in healthy individuals. Iron, lime, soda, magnesia, and the phosphates, are best

[1] Les differentes formes cliniques de la tuberculose pulmonaire, Paris, 1905, p. 157.

supplied by such foods as bread, flour, oatmeal, rice, sago, tapioca, fresh vegetables, and fruits. All these may be given plain, or, better still, in various other culinary preparations.

Condiments.—For their local appetizing effects, condiments, acting as they do as great salivary and gastric stimulants, may be taken, especially by those who suffer from anorexia. Some condiments, like mustard and garlic, contain allyl which assists in the digestion of fats, and is said to be bactericidal in the intestinal tract. At one time garlic was considered a good remedy against tuberculosis. Its active principle, allyl, was even administered subcutaneously.

Dangers of Overfeeding.—While the majority of patients stand a moderate increase in the quantity of food fairly well, there are many who are decidedly harmed by it. This is especially seen in those who have been unreasonably induced to increase the quantity of protein foods, such as eggs, meat, etc., thus imposing an excessive, and often dangerous, burden upon the liver, kidneys, etc. In some cases we find that these organs have been decidedly crippled by such a diet.

The symptoms produced by excessive protein consumption are unmistakable: The patient is drowsy for an hour or two after meals, has headache, and is irritable. At night he is restless and sleepless, or his sleep is disturbed by frightful dreams. The abdomen is distended, the liver enlarged, and may be tender on palpation. Heartburn, anorexia, bilious vomiting, and diarrhea often torture the patient. Cardiac palpitation and nightsweats are, at times, due to the indigestion thus induced. Because of the plethoric condition, these patients often have epistaxis, and also hemorrhoids, which contribute to their misery. The urine contains albumin, biliary pigments, indican, and glycosuria is not rare. Arthralgic pains in the joints are often the result of superalimentation. Older clinicians, observing that there exists an antagonism between the gouty and phthisical diatheses, urged excessive nitrogenous diet combined with wines, with a view of inducing sclerotic changes in the diseased lungs. On a similar principle, the excessive consumption of alcohol was advised in former days. According to some authors, the acneiform eruptions on the skin of some tuberculous patients are very frequently due to the excessive protein foods which they consume.

When overfeeding a patient we must watch out for the following danger signals: Failure of appetite, and symptoms of flatulent dyspepsia; dyspnea on exertion, which is obviously not due to the tuberculous toxemia or the lung lesion; diarrhea, and at times vomiting. If these symptoms are not heeded, and forced feeding is continued, irreparable damage may be done; the sheet-anchor of the patient, his power to digest food, is damaged, and his chances of recovery are materially lessened. But this should not deter us from trying to feed the tuberculous patient generously. "Excessive feeding is clearly a vastly better method of treatment than underfeeding, for it at least ensures the consumptive taking enough to repair his waste and to

restore his normal power of resistance and recuperation," say Bardswell and Chapman,[1] "The point to realize is, that it is quite an unnecessary hardship for patients to be overfed, and that it may do positive harm."

When these harmful results of unwise feeding are borne in mind, unfortunate patients will not be forced to ingest large quantities of food which may be excessive and dangerous to healthy persons. Especially careful must we be with plethoric, obese, and sedentary consumptives. A dilated stomach which does not empty itself with ease and promptness is particularly to be spared. The dangers of excessive ingestion of fat have already been dwelt upon.

Dietaries.—From what has been said, it is obvious that it is not necessary to give detailed dietaries for consumptives. When we aim at variety as the first requirement for a good diet, it would be necessary to give at least thirty menus to suit the average case. We will, therefore, merely mention some of the foods which may be utilized in attempts at feeding phthisical patients properly. It will be noted that they may eat nearly everything a healthy person can, so long as their malady is not complicated by conditions which alter matters.

Breakfast.—Milk, coffee, chocolate, cocoa, or tea. Bread, butter, cream, eggs, bacon, ham, ox tongue, fish (fresh or canned), fruit of any kind. Plenty of butter. Cereals of any kind.

Lunch.—Fish, or entrée; meats (roasts, chops, steaks, etc.), poultry, vegetables, custards, puddings, cheese, milk, coffee, fruit.

Dinner.—Soups, meats, poultry, game, fish, all vegetables, puddings, pastries, etc., cheese, ice-cream, coffee, milk or chocolate.

Without going into details of the various dishes that may be prepared by a good cook who knows the like and dislikes of the patient, it can be stated that there is no dish which is contraindicated in uncomplicated phthisis. *A good cook can do more for the patient than all the dietaries which may be printed in a book.*

Between the three main meals there may be allowed a light luncheon consisting of a glass of milk and some biscuit. Some are allowed an egg or two at that time, made in some form of punch, or in any style, provided it is well borne. Similarly, at about 4 P.M., tea, coffee, or milk may be allowed with some biscuit, etc. At night before retiring, a cup of milk with some crackers is beneficial for some patients. It will be noted that in this manner the patient may have his milk—about one-half to one quart per day—mainly outside of his meal-time, as drinks.

It must be emphasized again that these foods should be palatably prepared and rendered digestible by proper cooking. Otherwise trouble may arise. The quantity to be ingested depends on the personal equation of the patient, although in some cases matters may be forced for some time when indicated, but this should only be done bearing in mind the contraindications which have already been discussed.

[1] Diets in Tuberculosis, London, 1908, p. 49.

CHAPTER XLI.

MEDICINAL TREATMENT.

Importance of Medicinal Treatment.—The disrepute of medicinal substances in phthisis during recent years is due to several causes. The first and most important is that we have no specific botanical, chemical, or physical agent which, when administered to a consumptive, will exert a selective action on the tubercle bacilli, as mercury and arsphenamine do on the spirocheta of syphilis, and quinine on the malarial parasite. Nor have we a therapeutic agent which will enhance the resistance of the tissues against the ravages of the tubercle bacilli, or neutralize their poisons, or stimulate sclerosis of the affected area. But here we are in about the same position as when dealing with anemia, typhoid, pneumonia, rheumatism, etc. When we find that the salicylates relieve the most painful symptoms of rheumatism, and that iron increases the hemoglobin content of the erythrocytes in chlorosis, that digitalis increases the force of the cardiac muscle, we use these drugs although we know that digitalis does not regenerate destroyed heart valves, and salicylates do not remove the essential cause of acute articular rheumatism. Similarly if we find that creosote, arsenic, ichthyol, etc., have a beneficial influence on some of the annoying clinical phenomena of phthisis, though they do not cure the disease, we must not discard them merely because they do not remove the cause of tuberculosis, or kill the bacilli within the body, or neutralize the tuberculous poisons, etc.

There is another aspect to be considered in this connection. Excepting the chosen few, who have sufficient means to pay for first-class sanatorium treatment, and inclination to remain in the institution for months and perhaps years, the bulk of the patients must be treated in their homes. Even if they get a few months of sanatorium treatment in a public institution, they must be treated in dispensaries, or by their family physicians, before admission, and after discharge. The patient is a human being; and when we consider the human element we find that, as a rule, he has no confidence in a physician who has no remedy for his ailment. The dictum "plenty of fresh air, milk, and eggs," he believes he knows as well as the physician. If his medical adviser will not prescribe for him, he will seek remedies from another who is more obliging in this respect, or from an advertising quack. This is not only true of the ignorant, but also, almost to the same extent, of the supposedly intelligent patient.

It cannot be denied that in many respects medicaments, properly

administered, act by psychic suggestion. But so do the minute and detailed directions given, often in writing, about diet, rest, exercise, sleep, etc., in institutions. "Medicinal agents," says G. Küss,[1] one of the most ardent advocates of tuberculin treatment in France, "no matter in what they consist, always inspire confidence in the physician; without them he is helpless. Moreover, by giving the patient, in addition to other treatment, a prescription calling for some medicine, we may succeed better in our attempts at keeping him away from the alluring advertisements of charlatans who very often impose on him."

Harmless Medication.—The reasons why medicinal agents have fallen into disrepute in medical literature—by no means in the practice of the vast majority of physicians—are manifold. But the most important is perhaps the fact that drugs have been abused. "I regard medication as indispensable in the treatment of tuberculosis," says Rénon.[2] "It has an undoubted good effect on the disease in general and an enormous psychic effect. But there is one important condition which must be realized above all when giving drugs to consumptives—*they must be harmless.*" He illustrates this point by the following instance: Some years ago the acetate of thallium was suggested as an excellent remedy against the nightsweats of phthisis, and a trial showed that it did control this symptom very well indeed. But it also had another effect: It caused the hair to fall out, and the nails to shed. The patients stopped sweating, but incidentally lost their hair and nails, which was a good reason for resentment. That certain drugs used in phthisiotherapy may have disastrous effects in addition to their influence on the disease, or some of its symptoms, must always be borne in mind. In fact, it has been stated with considerable truth that 50 per cent of the dyspepsia in phthisical patients is due to improper medication.

Chemotherapy.—Ever since the bacterial origin of tuberculosis has been proved by Koch, attempts have been made to find a pharmacological agent which will destroy tubercle bacilli within the infected organism without simultaneously harming the host. Of course, the well-known antiseptics and bactericides inhibit the growth of the bacilli *in vitro*, and kill them in even great dilutions. But, for obvious reasons, the administration of these substances to animals or humans for therapeutic purposes is unthinkable. Indeed, it has been found that all substances known to inhibit the growth of tubercle bacilli in cultures fail to prove of therapeutic value in experimental tuberculosis in animals, and in spontaneous disease in human beings.

During recent years, since the great achievements of Ehrlich in the chemotherapy of diseases caused by protozoa, renewed attempts have been made to find a synthetic substance possessing a selective affinity for the cytoplasm of the tubercle bacillus without simulta-

[1] Gilbert and Carnot's Therapeutique, **21**, 594.
[2] Le traitement pratique de la tuberculose, Paris, 1908, p. 110.

neously harming the cells of the patient. But great stumbling blocks have been encountered in these attempts. To begin with the tubercle bacillus is surrounded by a fatty capsule, making it impermeable to the action of mild bactericides. Attempts at using lipolytic agents for the purpose of dissolving the waxy envelope of the tubercle bacillus have, so far, not met with encouraging results, especially in living organisms. Another hindrance to successful chemotherapy in tuberculosis is the fact that the tubercle is an avascular structure, as was shown when discussing the morbid anatomy of the disease. To reach the tuberculous lesions the remedy must be carried by the blood or lymph stream, irrespective of the route by which it is introduced into the body.

On the other hand, some authors have argued that avascularity may be altogether useful in chemotherapy by favoring the accumulation of the drug in tuberculous tissues. That this is feasible is seen in the fact that certain substances, as calcium salts, do enter tubercles and remain there. They are part of the natural mode of healing tuberculosis, and a therapeutic agent may likewise be brought into the diseased tissue. But, so far, no substance has been discovered which will penetrate the tuberculous tissues and remain there for a sufficiently long time to exercise beneficial effects.

It has been found by many authors, notably Wells and Hedenburg,[1] that the permeability of tuberculous tissue is like that of any simple colloid, permitting crystalloids to diffuse readily through them, but that it is hardly permeable to colloidal molecules. It has also been observed that tuberculous lymph glands take up relatively more iodine from the blood than other organs. Considering that iodine has been used for generations in phthisiotherapy, this was considered a hint in the direction of finding some synthetic compound of iodine which may prove of the same value in tuberculosis as arsenic, which also was used before the days of chemotherapy, in syphilis. But it was soon discovered that the deposition of iodine in tuberculous tissue is not entirely due to its selective affinity for the protoplasm of the tubercle bacillus, or the cells of tuberculous lesions, but that necrotic tissues caused by any other bacterial agent have the same proclivities, and that the amount of iodine found in necrotic tissue, tuberculous and others, depends mainly on a purely physical condition, the destruction of the semipermeability of the living cells.

An enormous amount of work has been done with a view of obviating these hindrances to attacking the tubercle bacilli in the infected organism, but so far no results worthy of the efforts has been attained. All attempts at destroying the fatty capsule of the tubercle bacillus through the agency of lypolitic substances have failed to show therapeutic results. The works of many indefatigable workers in Europe

[1] Jour. Infect. Dis., 1913, **11**, 349.

and in this country, among the latter may be mentioned Lewis,[1] Wells,[2] DeWitt,[3] and many others, using various elements, as copper, gold, iodine, etc., and various dyes, have so far not brought out a remedy which will cure or ameliorate experimental tuberculosis in animals, or spontaneous disease in man. Apparently the reason for this failure of chemotherapy is to be sought in the complex etiology of the disease. To be sure there is no tuberculous disease without tubercle bacilli, but as we have already shown (see Chapters IV and V), infection is followed by disease only in a comparatively small proportion of persons into whose bodies these bacilli have gained an entrance. It seems that a specific remedy will have to be directed against certain constitutional defects which render the body vulnerable and which we do not understand clearly at present, and not alone against the tubercle bacillus. But even here we are not on sure ground at the present state of our knowledge of phthisiogenesis.

One thing must be emphasized in this connection. Many who have some drug which they believe effective in relieving some of the symptoms of tuberculous disease do not hesitate to call it chemotherapy. In a certain sense this may be considered correct. But, as defined by those who work along these lines, chemotherapy consists in the introduction into the body of bacteriotropic substances which are not in the same degree organotropic; substances which though very harmful to the virus of the disease are hardly harmful, if at all, to the cells of the host. Such a substance is, at present, an ideal, a hope of many indefatigable workers.

However, though no specific remedy has as yet been found for tuberculosis, there are many which are of immense utility in our efforts at relieving the symptoms which torture the patient. Some of these will be discussed here.

Creosote.—There are but few sufferers from tuberculosis who have not been given creosote at some period of their illness. Its history is similar to that of tuberculin. Introduced by Reichenbach, in 1830, it was given in very large doses, resulting in considerable harm to the patients. It was discarded for this reason, to be reintroduced some thirty years ago, and ever since it has held its place in the armamentarium of the physician in general and special practice. Its most ardent advocates do not consider it a specific, but then those urging tuberculin are still looking for a specific for tuberculosis. In the hands of those who have administered it intelligently it has proved the best medicinal agent to relieve some of the most baneful symptoms of the disease.

Those favoring this drug claim that when administered in the proper cases, and in proper dosage, it improves the appetite, stimulates digestion and assimilation, improves nutrition, diminishes expectoration,

[1] Johns Hopkins Hosp. Bull., 1917, **28**, 120.
[2] Jour. Infect. Dis., 1913, **11**, 349.
[3] Ibid., 1913, **12**, 68; **13**, 378; **22**, 426.

removing, at times, its purulent character and disagreeable taste and odor, all of which are sufficient encouragement to the average sufferer from phthisis to bestow confidence in the physician, and to look forward to an ultimate recovery. Some earlier writers were inclined to ascribe this beneficial action of creosote to its power to inhibit the growth of, or destroy, tubercle bacilli in the gastro-intestinal tract, which are inevitably swallowed by every consumptive. For obvious reasons this is absurd. But it is a fact that it is one of the best gastric and intestinal antiseptics we have. It has been found that part of the ingested drug is excreted by the bronchial mucous membrane and, while it cannot be expected to destroy the bacilli in the lungs—hardly any drug could reach the avascular tubercle, even if it could be given in sufficiently large doses—it is said to exert there a beneficial influence, as is evidenced by the decrease in the amount of sputum brought out, and the diminution in the intensity of the associated bronchitis, laryngitis, and tracheitis. Pharmacological evidence along these lines is, however, wanting. Experimental tuberculosis is not at all influenced beneficially by creosote. Lydia M. DeWitt[1] and her coworkers have even found its bactericidal power very low.

It is a peculiar fact, not generally appreciated, that creosote often provokes general and local reactions which are analogous to those provoked by tuberculin. Usually with excessive doses, but occasionally also with minimal doses, after taking creosote for several days the patient is overtaken by a feeling of chilliness and fever, pain in limbs, back and joints, weakness, fatigue, and insomnia. Malaise, gastric disturbances and even vomiting in patients whose stomachs have heretofore not given any trouble, now make their appearance. The part of the creosote eliminated through the bronchial mucous membrane often excites a focal reaction which, at times, reminds one of the focal reaction of tuberculin. Of course, in the case of tuberculin a single dose is often enough to produce this reaction, while in the case of creosote it is only the more or less prolonged administration that is apt to produce this effect. In such cases sanguineous expectoration and even hemorrhage are not uncommon, while the lesion in the lung may be aggravated, or even spread. Rales, which were previously absent or scanty, now make their appearance and the general aspect of the patient is aggravated.

If the administration of creosote is persisted in after these symptoms manifest themselves, the condition of the patient may be aggravated to an extent as to render the prognosis hopeless in a case that previously had a fair outlook. Smoky urine, like that of phenol poisoning, is now seen; the patient complains of a taste of creosote in his mouth. This may be followed by vertigo, profuse perspiration, chilly sensations, and even cyanosis and collapse, as I have seen in one case which was greatly relieved by the discontinuance of the drug.

[1] Jour. Infect. Dis., 1920, **27**, 115.

Contraindications.—Bearing all this in mind we can say that *creosote is contraindicated in all cases in which it provokes gastric disturbances.* If after taking moderate doses of the drug the appetite does not improve, it should be discontinued. It is also contraindicated in all febrile cases in which the temperature is 100° F. or more, and also in all progressive cases, because they are the ones in which general and local reactions are apt to be provoked and spread the lesion in the lungs.

Patients subject to hemoptysis must not be given any creosote; even blood-streaked sputum should serve as a warning for the immediate discontinuance of the drug. Moreover, one must not wait for the appearance of smoky urine, but carefully watch for albumin which is often brought about by creosote. In general, albuminuria is a strong contraindication to the administration of creosote.

Indications.—*In all incipient cases in which the appetite is poor and digestion defective, creosote may be given.* With the improvement in the nutrition of the patient, owing to cessation of gastric and intestinal fermentation, the local condition in the lungs also shows improvement. In chronic, sluggish, afebrile cases of tuberculosis, especially those characterized by profuse expectoration, creosote is often of immense benefit, if rationally administered. In addition to its good effects on the gastro-intestinal functions, it also diminishes the amount of expectoration, ameliorates the cough, etc., and with the gain in weight and comfort, it has an excellent effect on the psychic state of the patient, who becomes more encouraged and hopeful. In fibroid phthisis, characterized by profuse expectoration of purulent material, provided there is no concomitant emphysema, next to the iodides, creosote is the best remedy we have.

Administration.—A good product must be used. Soon after its introduction creosote fell into disuse mainly because of the bad quality of the product. Good creosote, fit for therapeutic administration, must be obtained from the fractional distillation of beech-wood tar. The product dispensed in many pharmacies in this country is obtained from the distillation of bituminous coal, and contains many impurities which are not well tolerated. A good preparation of creosote contains 25 per cent of guaiacol, but many of the products dispensed under this name, even when obtained from beech-wood, contain much less.

It is best administered in capsules which do away with the disagreeable odor. Moreover, the mucous membrane of the stomach and intestines is not so easily injured by creosote as that of the mouth and pharynx, so that the disagreeable local effects are done away with through capsules. Some mix it with balsam of tolu, and it is best given after meals. Those who cannot swallow capsules may take it in this form:

℞—Creosoti	gtt. xxx	2.0
Vini pepsini	℥iv	120.0

M. S.—Teaspoonful in water three times a day after meals, gradually increasing.

℞—Creosoti,
Picis liquidæ radicis . . . . . ää gr. xxiv 1.5
Alcoholis absol. . . . . ʒiij 12.0
Balsam. peruv. . . . . ʒiv 15.0
Tinct. Helianthi annui . . . . . ʒv 20.0
Olei terebinth. rectificati.
Myrtholi . . . . . ää ʒij 7.5
M. S.—Three times a day, one teaspoonful in milk or water one hour after meals.

℞—Tannini . . . . . ʒv 20.0
Calcii phosphorici . . . . . ʒv 20.0
Creosoti . . . . . ʒiiss 10.0
M.—Div. in part 40; ft. capsul.
S.—One capsule three times a day after meals.

Beverley Robinson has had good results with the following:

℞—Creosoti . . . . . gtt. vj 0.5
Glycerini . . . . . ʒj 25.0
Spiritus frumenti . . . . . ad ʒiij 100.0
M. S.—Teaspoonful in water three times a day after meals.

This dose may be increased to two or three teaspoonfuls, or, if it is desired to increase the creosote, the amount of it may be doubled. If the whisky is deemed inadvisable, elixir calisaya or the compound tincture of cardamom may be substituted.

Many have administered creosote by inhalation and have obtained good results. In this country, Beverley Robinson introduced this method. He recommends equal parts of creosote and alcohol or, when there is much irritative cough, equal parts of creosote, alcohol, and spirits of chloroform, on the sponge of a perforated zinc inhaler. The inhaler should be used frequently, at first for a few minutes, later gradually increasing the time until it is used from half an hour to an hour at a time, and finally it may be used almost continually during the day and frequently all night. These inhalations have a salutary psychic effect on some patients who feel that something is being done for them and the odor of the substance impresses even those around the patient that an effective remedy is administered.

The following are good formulæ for inhalation:

℞—Creosoti . . . . . gtt. vij 0.5
Tincturæ benzoini comp. . . . . ʒiij 100.0
M. S.—To inhale a teaspoonful from boiling water, three or four times a day; shake.

℞—Creosoti . . . . . gtt. vij 0.5
Olei pini silvestris . . . . . ʒiiss 10.0
Olei terebinthinæ . . . . . ʒiss 5.0
Tincturæ benzoini comp. . . . . ʒiv 100.0
M. S.—Shake. To inhale a teaspoonful from boiling water, three or four times a day.

Derivatives of Creosote—Because of its caustic taste, and disagreeable odor, creosote is not well tolerated by many patients; even when given in capsules the odor is often penetrating. Guaiacol, the main active principle of creosote, can be given instead, but it is insoluble in water, has an objectionable odor and taste and is a gastric irritant.

There have been brought out a large number of preparations which retain most, or all, of the useful qualities of creosote without its drawbacks. These derivatives of creosote are mostly used at present with the same result as with the original drug.

Of these creosote carbonate (creosotal) is perhaps the best. When ingested it breaks up slowly in the intestine, liberating creosote. It may be given in capsules of 5 to 10 drops three or four times a day. Many pharmaceutical houses market globules which are very elegant. It may also be given to patients to be taken in a certain number of drops in water, milk, or coffee; or the following prescription is useful:

℞—Creosoti carbonatis	ʒiv	120.0
Ætheris	ʒiss	5.0
Alcoholis sol.	ʒvj	25.0
Vanilin	gtt. vij	0.5

M. S.—Fifteen drops in water or in milk three times a day after meals; increased if well tolerated.

In many cases between 30 and 60 grains of creosote carbonate may be given per day. Guaiacol carbonate (duotal) is another preparation which is very extensively used. It may be given in powder or capsule from 10 to 30 grains a day, or combined with arsenic.

Both of the above preparations are now sold quite reasonably. But for those who can afford to pay, we have a wider range of choice. Styracol (guaiacol cinnamate) contains a high percentage of guaiacol. Thiocol (potassium-guaiacol-sulphonate) may be given in 5 to 15 grains three times a day in powder, tablet, or capsule. It is a non-toxic, tasteless, odorless powder, soluble in water. Many patients who do not tolerate guaiacol take this preparation very well, and in those who suffer from diarrhea it is to be preferred. But it contains less guaiacol than most other preparations of this class and its action is not so intense as that of the others. In fact, it is sometimes not decomposed in the intestines, and may be excreted unchanged. For those who prefer their medicine in liquid form and for children, it may be given in the form of sirolin, a 10 per cent solution of thiocol in orange syrup, which may be given one to three teaspoonfuls three times a day. There is no doubt that many who cannot tolerate creosote or guaiacol take this less toxic preparation very well.

Sir R. Douglas Powell recommends the following:

℞—Guaiacol carbonatis, guaiacol benzoatis vel styracol	ʒiss	6.0
Calcii hypophosphatis	ʒss	2.0
Pulvis tragacanthæ comp.	ʒj	4.0
Misce bene, adde guttatim:		
Syr. pruni virginianæ vel elixir aurantii	ʒss	16.0
Syr. calcii lactophosphatis vel syr. hypophosphitum comp.	ʒj	32.0
Aquæ chloroformi ad	ʒvj	190.0

S.—One teaspoonful in water or liquid malt three times a day soon after meals.

℞—Creosoti carbonatis	ʒiv	16.0
Tinct. gentianæ comp.	ʒiv	16.0
Syr. pruni virginianæ	ʒiij	90.0

S.—One teaspoonful in a wineglass of water or malt extract after meals three times a day. Increase the dose by five drops each second day up to two teaspoonfuls by measure.

Ichthyol.—Ammonium sulphoichthyolate or ichthyol has been found very useful in many cases of phthisis. Some authors state that it has a favorable influence on the metabolism, prevents albuminous decomposition and favors assimilation of food. Helmers found that about one-third of the sulphur ingested with ichthyol circulates in the juices of the body; others asserted that it even has a bactericidal action, without hurting the body cells, etc. It may, however, be stated that we do not know the exact pharmacology of this preparation, but that empirically it has been found useful in many cases of phthisis.

It may be given in water 2 to 5 drops three times a day, beginning with the smaller dose and gradually increasing according to tolerance. Because of its disagreeable odor and taste, the drops should be diluted in large quantities of water or milk and given before meals. It may also be administered in black coffee. Or the following formulæ may be used:

℞—Ichthyolis		ʒvj	25.0
Aquæ distil.		ʒij	60.0
Alcoholis rectific.		ʒij	60.0
Syr. citr.,			
Syr. aurant cort	āā	ʒiss	50.0

M. S.—Teaspoonful in water three times a day before meals.

De Renzi says that the above formula conceals the taste and odor of ichthyol. The following is also of use:

℞—Ichthyolis		ʒiiss	10.0
Syrup. simpl.		ʒv	20.0
Aquæ menth. piper.		ʒiij	80.0

M. S.—Teaspoonful in a glass of water three times a day.

In many cases ichthyol improves the appetite, diminishes the frequency of the cough and the expectoration, changing the latter so that its purulent character vanishes. The general condition of the patient improves with the improvement in the nutrition. In some patients the remedy disagrees, causing flatulence, abdominal pains, diarrhea, loss of appetite, and eructation of gases. In fact, as has been shown by Barnes, in patients in whom the administration of ichthyol does not immediately improve the appetite, it is not advisable to continue the drug. I can add that diarrhea also shows that the drug disagrees. My patients do not, as a rule, mind the disagreeable odor and taste when given well diluted with water, milk, or coffee.

Ichthyol should be tried in every case of phthisis because it has not the dangerous characters of creosote and arsenic and their derivatives; in fact, it is well tolerated in most cases, only gastro-intestinal disturbances occasionally preventing its use.

Arsenic.—For centuries arsenic has been used by physicians in the treatment of tuberculosis. As has been pointed out by A. Arkin and H. J. Corper,[1] Dioscorides employed it internally and by inhalation.

[1] Jour. Infect. Dis., 1916, **18**, 335.

Antylus, who lived in the third century A.D., Marcellus Empyricus, and Galen all recommended it and described cures from the inhalation of powdered arsenic. The Chinese and the Hindus also found it useful in tuberculosis. Empirically, it has also been employed by modern physicians in various forms, and many report excellent results. While some claimed that it has a direct action on the tubercle bacilli, recent careful investigations by Arkin and Corper have shown that this is not the case. Many preparations of arsenic—sodium arsenite, sodium cacodylate, mercury cacodylate, atoxyl, arsacetin, and neoarsphenamine have all been found without any action on tubercle bacilli *in vitro*. Administered to tuberculous animals parenterally these preparations of arsenic were subsequently found in the liver, lungs, kidneys, blood, spleen, and tuberculous tissues (lymph glands of guinea-pigs and eye of rabbit), the concentrations in all these tissues not greatly differing. No evidence of accumulation in the tuberculous tissues was obtained thus showing that it has no selective affinity for tubercle.

Clinical experience has, however, shown that arsenic is an excellent stimulant of nutrition, a hematinic, reconstructive, and alterative in chronic wasting diseases, including phthisis. The various organic arsenic compounds recently introduced were stated to lack the greater part of the toxicity of arsenic, while retaining its curative, reconstructive, and antiseptic properties. The advocates of arsenic medication in tuberculosis claim that it increases the appetite, improves assimilation of food, and stimulates the blood-forming organs, in addition to its stimulating effects on the nervous system. In short, arsenic is supposed to fortify the tissues against the ravages of the tubercle bacilli.

From an extensive use of arsenic in phthisis the author has not found that it exerts any direct influence on the tuberculous lesion in the lungs, even when administered to patients who tolerate it. The quantity and quality of the expectoration are, however, very favorably influenced in some cases; purulent sputum often becoming mucous and greatly reduced in quantity. With the improvement in the appetite and nutrition a great deal is gained—the patient is encouraged. The fever is, however, not influenced, nor are the nightsweats. In fact, it should not be given to febrile patients.

It may be given as an adjuvant to creosote treatment in the form of trioxide, as in the following formula:

℞—Guaiacolis carbonatis	ʒv	20.0
Arsenici trioxidi	gr. iss	0.1
Strychninæ sulphatis	gr. j	0.06

M.—Ft. pilulæ no. lx div.
S.—One pill three times a day after meals.

It may be given in the form of Fowler's solution, beginning with 2 or 3 drops after meals and increasing daily until 10 drops are taken three times a day.

During recent years various organic compounds of arsenic have been used in phthisis, administered either by mouth or hypodermically.

Of these the cacodylates of sodium, strychnine, iron, and guaiacol may be mentioned. Many of these, as well as atoxyl, are at present sold by pharmaceutical houses in ampoules ready for hypodermic and intravenous administration. But in my experience none of these preparations has any advantages over the inorganic arsenic; the trioxide, and Fowler's solution, answer all requirements. In fact, some of them, notably atoxyl, are dangerous because they are liable to cause amblyopia. Attempts at utilizing arsphenamine in the treatment of phthisis have met with failure to observe beneficial therapeutic effects, excepting in tuberculous patients who also have syphilitic lesions.

When administering arsenic to phthisical patients certain precautions are to be taken. It should not be continued, especially in large doses, for more than a week or ten days. Symptoms of intolerance may make their appearance, such as loss of appetite, thirst, and dryness in the mouth, colicky pains, and diarrhea. In some cases the fever rises as a result of large, or even small, doses of arsenic. Tachycardia, cardiac palpitation, and insomnia are occasionally observed. It should not be given to febrile patients, and to those showing a tendency to hemoptysis. In fact, if during the administration of arsenic there appears streaky sputum, it should be considered a danger signal and the arsenic is to be discontinued at once.

Iodine.—For generations iodine has been used in the treatment of scrofulous children with good results. It has also been found useful in assisting the resolution of pleural adhesions, and in the relief of the symptoms of chronic bronchitis, pulmonary emphysema, and asthma. That the iodides have an effect on tuberculous lesions in the lungs is evidenced by the fact that small doses of the iodide of potassium may cause, in persons with incipient tuberculosis, reactions similar to those produced by tuberculin, as was shown by Rondot. In fact, many authors recommend it for diagnostic purposes, at least to provoke expectoration which may be examined for tubercle bacilli. Sorel[1] found that tuberculous animals, when given large doses of potassium iodide, succumb to generalized miliary tuberculosis, and usually much earlier than the controls.

Recent investigations tend to show that iodine counteracts and inhibits the lipoid element in the tubercle bacilli. Joblings and Petersen found that soaps of the unsaturated fatty acids were capable of inhibiting the action of trypsin and other ferments, and, moreover, they discovered in the tubercle bacilli a ferment inhibiting substance of the nature of a lipoid, to which they attribute the lack of autolysis and consequent caseation in tuberculosis. They found that the higher the iodine value of a soap the less was its activity as an inhibiting agent, while saturation with iodine would destroy entirely its inhibiting powers. They also found that ether-soluble substances of the bacilli,

[1] Ann. de l'Inst. Pasteur, 1909, **23**, 533.

which constitute 25 to 35 per cent of their weight, and which are largely composed of fatty acids, have a marked restraining action on trypsin. It is thus suggested by E. Curtin that iodine acts in tuberculosis by saturating the unsaturated bonds of the fatty acids of the lipoids, rendering the substituted product less active as an antitryptic agent.

Some French authors recommend the iodides in most cases of pulmonary tuberculosis, but it seems to be a dangerous drug for the reasons just stated. But in some cases of incipient phthisis without fever the iodides do good, especially in those in whom the tuberculous process has been implanted on emphysematous lungs. This is also true of asthma and tuberculosis—the iodides often control or relieve the nocturnal attacks of dyspnea. But one must always guard against giving this drug to sufferers from the congestive, inflammatory, progressive lesions, and those subject to hemoptysis.

It is best given in a saturated solution of iodide of potassium of which each drop represents 1 grain of the drug. Small doses are to be given at first, 2 to 5 grains, three to five times a day. If no intolerance is shown it may be increased. I have often used some of the organic compounds of iodine—sajodin, etc.—with good results.

A better way of administering iodine is giving the pharmacopeial tincture in increasing doses, beginning with one drop well diluted in water or milk, three times a day, and increasing daily by one drop, until twenty or even thirty drops are given daily, or until toleration is reached. Some patients show symptoms of iodism very soon, and the dose must be reduced, but in the majority of cases large doses may thus be given for a long period with very marked results. In fibroid phthisis it has often proved invaluable.

Mercury.—Mercury has been used in the treatment of tuberculosis for many years. But more recently Dr. B. L. Wright developed a new method of administering it and reported a larger number of recoveries than has been claimed with any other medication. He used the succinimide of mercury hypodermically, in doses of $\frac{1}{5}$ of a grain given on alternate days, increasing the dose guardedly until the limits of toleration are reached. As soon as symptoms of mercurialization appear, or there is a rise in the temperature, anorexia, loss in weight, etc., the dose is either reduced or the treatment is discontinued for a time. In most cases about thirty injections are given, followed by a rest of two weeks, during which period iodide of potassium may be administered. A second series of injections is given to those who tolerate the drug.

I have tried this treatment and found it of immense value in phthisis complicating syphilis; otherwise it is decidedly harmful. As was already stated, it appears that when tuberculosis is implanted in a syphilitic subject, the disease is apt to run a very sluggish, chronic course. Fibrosis is very active. In these cases both the iodides and mercury, if intelligently and guardedly administered, may be very

efficacious. The succinimide of mercury may be used instead of other forms of the drug. But the doses given by Wright are decidedly excessive—the same results may be obtained by the hypodermic administration of $\frac{1}{8}$ or $\frac{1}{12}$ of a grain twice weekly. On the other hand, arsphenamine now offers a better means of combating active syphilis combined with tuberculosis than the succinimide of mercury.

Hypophosphites and Glycerophosphates.—It will be noted that most of the medicinal preparations mentioned above have their indications and contraindications, and some are not without danger when improperly administered. The safest medication in phthisis appears to be the time-honored administration of the hypophosphites. Recently the glycerophosphates of lime, iron, magnesium, etc., have been used very extensively on the theory that phthisis is a manifestation of lime starvation and that recalcification and remineralization of the body are of great importance in our efforts at combating the effects of the tuberculous process. There is no doubt that in many cases of phthisis these medicinal substances have an excellent influence on the nutrition of the patient and they are also of use in relieving the anemia which is such a frequent accompaniment of the disease. We may give the official compound syrup of hypophosphites in doses of one to two teaspoonfuls three times a day after meals. The glycerophosphates may be given in any form. Pharmaceutical houses have many elegant and palatable preparations of glycerophosphates in tablet, capsule, and liquid forms which may be used. Their onic effects are beyond question.

Cod-liver Oil.—Physicians of past generations bestowed great confidence in the therapeutic virtues of cod-liver oil in tuberculosis, and many modern practitioners still consider it an excellent therapeutic agent. Some have ascribed the curative action of this oil to certain of its constituents. Thus, some believe that it is the iodine which is effective, others see in the bromine the active principle. But careful chemical analysis has shown that there are only traces of these elements in cod-liver oil. The biliary salts, the hepatic ferments, the lipoids, the lecithin, etc., have been stated to be of more value than the fat of cod-liver oil. John W. Wells,[1] and others believe that, in addition to the ready absorption of the fat of cod-liver oil, it possesses powers of increasing the absorption of other fats of the food to a marked degree.

The recent intensive studies of the internal secretions have also thrown some light on the action of cod-liver oil in phthisis, according to some authors. Thus, Williams[2] stated that the superiority of this oil to others is mainly due to the internal secretion of the liver of the fish, which "when introduced into the human economy, acts as a stimulant to one of the normal internal secretory glands, and the secretion of the one so stimulated is inimical to the development of the tubercle bacilli." He believes that only the crude oil contains

[1] British Med. Jour., 1902, **2**, 1222.
[2] Practitioner, 1911, **88**, 605.

these active principles and is therefore more efficacious than the refined oil. Iscovesco,[1] from his experimental researches, is convinced that the efficaciousness of cod-liver oil is due to the lecithides which it contains. He treated a large series of animals for four months. Those which got cod-liver oil increased in weight to the extent of 55 per cent; those which got cod-liver oil from which the lecithides had been removed gained only 27 per cent; those who were given olive oil gained 33 per cent; others were given oil to which was added 0.5 *pro mille* of the lecithides extracted from cod-liver oil and they gained 56 per cent. The control animals gained only 29 per cent. Williams and Forsyth[2] claim that the unsaturated fatty acids of cod-liver oil tend to disintegrate the waxy envelope of the tubercle bacilli, thus destroying them.

These theories are interesting, and deserve further study, but there is no doubt that cod-liver oil is an important remedy in tuberculosis, even if only for the fact that it contains a considerable proportion of easily assimilable fat, and it may be used as a food rather than as a drug. Patients who do not take such animal fats as butter, etc., are distinctly benefited by cod-liver oil.

Cod-liver oil should be given in large doses; to some patients as much as 2 ounces per day may be given and some French authors, like Jaccound, Grancher, and Daremberg, have given more than 4 ounces per day. Some apparently have a marked tolerance for this preparation, and they may utilize it instead of superalimentation. On the other hand, there are patients who cannot tolerate it, and even small doses cause eructations, nausea, and oily taste in the mouth. Diarrhea is another of the untoward effects in some who do not bear the oil very well.

Indications.—Cod-liver oil is indicated in all afebrile cases of phthisis. All patients who willingly take it and digest it well in large doses should be given this oil, without incidentally curtailing their usual amount of other nourishment. It may be continued for a long period of time; as long as the patient is apparently benefited by it and his digestive functions remain normal, the appetite is good and, above all, there is no diarrhea. Patients with fever do not tolerate it as well as those who have no pyrexia. Children with tendencies to scrofula, with enlarged tuberculous glands, especially tracheobronchial adenopathy, and who are as a result underfed and anemic, often derive great benefit from cod-liver oil. It appears that children take it with greater ease, and more often with distinct benefit, than adults.

Contraindications.—Cod-liver oil is contraindicated in cases in which the patients do not tolerate it in even small doses. The best criteria are the state of the appetite and digestion. As soon as these are deranged, it should be discontinued.

[1] Compt. rend. Soc. de biol., 1914, **76**, 34.
[2] British Med. Jour., 1909, **2**, 1120.

Administration.—So long as we consider cod-liver oil merely a fat food, and disregard its other constituents, it is best to administer it in as palatable a form as possible. In former times the crude oil, a product of decomposition of the livers of the cod, was used. Some modern authors even now insist that this form is most beneficial for phthisical patients. But it has a very disagreeable odor and taste and it requires courage on the part of the patient to swallow it. It is also apt to cause indigestion, eructations, diarrhea, etc. The light, or amber-colored oil, prepared by melting fresh livers by a steam process, is less disagreeable and more easily tolerated. It should at first be given in small doses of the Norwegian, light-colored oil, and in case the gastro-intestinal tract tolerates it, the dose is to be increased so that within a few weeks the patient takes four to six tablespoonfuls a day after meals. It should not be forced on patients; when they refuse to take it, or if it causes nausea, eructations, diarrhea, etc., it should be discontinued.

It is best that the pure oil should be given and many patients take it easily. With some the odor and taste have to be masked, and this may be done in the following manner: It may be given in orange-juice, or in some volatile oil. Many patients take it with ease in coffee or milk. A pinch of salt placed in the mouth before taking it may disguise the taste. Those who are allowed to take alcohol may take some whisky or brandy into the mouth where it is kept for a few seconds without swallowing, and then the oil is taken. Some use peppermint-water or tomato ketchup for the purpose, or orange- or lemon-juice. The difficulties owing to the odor and taste are overcome soon in most patients, and they take it freely.

The various emulsions offer no advantage over the pure oil. If they contain the indicated percentage of the oil, they are as disagreeable as the pure article, and one who can take an emulsion can take and digest the oil. The various preparations and "extracts" which are alleged to have all the therapeutic qualities of cod-liver oil without any of its disadvantages, have been found worthless, lacking as they do the fatty substances which are of value for the nutrition of the patient. On the other hand, many of the preparations of cod-liver oil and malt, hypophosphites, creosote, etc., may be utilized in the treatment of phthisis with advantage. It is, however, to be borne in mind that large doses are necessary to procure results, and that these preparations contain but a small proportion of cod-liver oil.

CHAPTER XLII.

SPECIFIC TREATMENT.

STRICTLY speaking, the term "specific" should only be applied to a remedy or preparation which has a proved selective curative effect on a certain disease. From this viewpoint *we can state unequivocally that we have no specific remedy for tuberculosis in any of its clinical forms*. We have no substance, drug, or preparation which will cure, or remove, or ameliorate the symptoms in the vast majority of phthisical patients to the same degree as mercury or arsphenamine is efficacious in syphilis, quinine in malaria, or thyroid in myxedema. This is a fact which all thoughtful workers in the tuberculosis field acknowledge; even those who employ tuberculin extensively, and do not hesitate to call it specific treatment, say that it is only a good adjuvant to other therapeutic methods which should be tried in selected cases so long as a true specific is not available. Moreover, it appears that tuberculin only works in sanatoriums, where the patients are, in addition to the specific treatment, subjected to a rigorous hygienic and dietetic regimen. It is distinctly stated that when the latter is lacking, tuberculin is of no avail.

It appears that the only justification for the use of the term specific when speaking of tuberculin treatment is the fact that this word has recently received a wider application and is now also used to designate remedies which are especially indicated, and used, in any particular disease.

The writer has given tuberculin therapy a fair trial in both his hospital and private practice and found it either altogether wanting in therapeutic effects when used in infinitesimally small doses, as is advised by most of its contemporary advocates, or decidedly harmful when given in substantial doses. This opinion is shared by most of those engaged in the treatment of tuberculosis, excepting such as have themselves discovered some tuberculin, or who are in charge of sanatoriums catering to well-to-do private patients. In the public sanatoriums in this country very little of tuberculin is used for therapeutic purposes. The vast majority of patients in these institutions are cared for by the old methods. It cannot be said that it is the cost which precludes the use of tuberculin in public institutions. Arsphenamine is a really expensive drug but is used in all hospitals.

Our reasons for discarding tuberculin from the therapeutic armamentarium are the following:

The Variety of Tuberculins.—It is an old axiom in therapeutics that the larger the number of drugs recommended for any given disease, the less the chances of curing it with any of those mentioned as effica-

cious. Thus, we have only to consult the index of any standard materia medica and count the number of remedies recommended for typhoid fever, pneumonia, nephritis, gastritis, etc., and to compare it with the number mentioned as effective in myxedema, malaria, syphilis, valvular heart disease, etc., to be convinced that the axiom holds good. The large number of tuberculins alone should give us a strong hint that none of them is a specific, or will surely cure. I counted in one German book forty-six varieties of tuberculins, and I could add almost as many which the author has not mentioned.

"We have no standard tuberculin," says William Charles White,[1] himself an advocate of tuberculin, "and furthermore we have no manufacturer who prepares the same strength twice. Consequently the dose of one tuberculin is no more the dose of another tuberculin than the dose of a sherry glass is the dose of a champagne glass. We have no method of testing the strength of a given tuberculin unless it is the biological one, and this is tedious, if it has to be used for every patient for every new supply of tuberculin. If, however, the tuberculin standard is at fault, what a vastly greater difference exists in the physicians who administer it? There are almost as many methods of dosage and administration as there are administrators. Each physician believes his method the best. Some have no method at all." It appears that for practical purposes we have no methods to weigh or measure the toxicity of tuberculins. Two preparations made by the identical method may differ very much if they are derived from different cultures; especially do they vary with the age of the culture.

All authors entitled to an opinion agree that the action of all tuberculins is the same. The preparations differ only as regards their strength, toxicity, capacity for absorption, etc. But inasmuch as the active element or substance of tuberculin has not yet been isolated, nor can the strength of a given preparation be measured, it appears that the differences which are known to exist between the various forms of tuberculin cannot be definitely ascertained. Arsphenamine strychnine, morphine, digitalis, or tetanus and diphtheria antitoxin which could not be measured would hardly be used by medical men.

In general it may be stated that there are three varieties or types of tuberculin:

1. *Old tuberculin*, consisting of the exotoxin—a glycerin extract containing the soluble products of the tubercle bacilli in the medium in which they have grown, glycerin, bouillon, extractives, etc. Though it should be mentioned that most investigators are of the opinion that there is no tuberculous exotoxin.

2. The *new tuberculins*, made up of the insoluble endoplasm of the bacilli and the poisons contained within them—endotoxins.

3. Those which consist in a mixture of both the above forms.

[1] Tr. Fifth Annual Conference Nat. Assn. Prevent. Consumption, London, 1913, p. 70.

But when injected into the tuberculous human or animal body any tuberculin produces practically the same effect. On this nearly all agree, even those who maintain that only a certain variety of tuberculin should be used if therapeutic results are to be obtained.

Action of Tuberculin.—As was already stated (see p. 37), tuberculin is harmless when injected into a non-tuberculous body, and produces its toxic effects only in those who have suffered a tuberculous infection. But we do not know how it acts under these circumstances. Wolff-Eisner's tuberculolysin hypothesis is about the most plausible and the one accepted by most authors. But we have not as yet succeeded in isolating a specific tuberculous antibody, nor the tuberculolysin from the serum of infected animals.

At first sight it would appear that tuberculin is specific, considering that it acts only on infected organisms, but even this is not conclusive. It seems that the infected organism is not only hypersensitive to tuberculin, but to all foreign proteins. We can produce elevation of temperature, malaise, backache, nausea, etc., and even the local reaction, by the injection of any foreign protein into a tuberculous person. "Neither the local nor the general reaction is absolutely specific," says Baldwin,[1] himself using tuberculin extensively; "various nucleoproteins, yeast nuclein, bacterial proteids in general, and digestive products, such as albumoses, are capable of producing similar effects. Cinnamic acid, cantharidin, pilocarpin, and other alkaloids also act to some degree, although less as local irritants than general leukocyte stimulants." Parenteral milk injections have also been found to produce general and local effects not unlike those of tuberculin. In my experience, potassium iodide and creosote, when given in large doses, may produce general and focal reactions not unlike those produced by tuberculin.

All efforts at producing partial or complete immunity with the administration of tuberculin in man or animals have utterly failed. Even Sahli, who urges tuberculin treatment, says that "tuberculin treatment has not the character of a true immunization, though it produces immunizatory effects in the organism."

That it is not necessarily the reaction which is effective therapeutically is clear when we consider that modern tuberculin treatment aims at eliminating entirely these reactions by the administration of infinitesimally small doses. The hope that the focal reactions, consisting in hyperemia at the site of the lesion, and the surrounding tissues, may promote the healing of the lesion, cannot be seriously entertained by clinicians. Usually when the focal reaction is intense, it cannot be controlled and the congestion often produces renewed activity of the diseased process. Quiescent foci, calcareous particles, are "sleeping dogs" and should not be disturbed, as Sir James K. Fowler[2] says. The establishment of tuberculin tolerance, which some

[1] Osler's Modern Medicine, **1**, 308.

[2] Tr. Annual Conference Nat. Assn. Prevent. Consumption, London, 1913, **5**, 93.

strive at, is no proof of healing; in fact, it is usually short-lived. Moreover, the tuberculin reaction is a very complex process and varies with the preparation used, the individual treated and also with the time it is administered. One day the patient is tolerant, the other he is badly affected with even a minimal dose.

There is no harm in administering most drugs in teaspoonfuls, tablespoonfuls, or measuring them with the point of a knife, as has been done for centuries. Patients have recovered with such inexact measures, some may have been harmed, but lethal doses are rarely given in this manner. But we cannot give a potent agent like tuberculin to a patient who needs all the vital energy he has, and more, in this manner, any more than we can give with impunity strychnine, morphine, digitalis, arsphenamine, etc., without exact dosage. So long as we cannot measure the toxicity of tuberculin, we cannot administer it rationally and prevent sudden and at times harmful, reactions which may appear when least expected.

Experimental Evidence of the Lack of Therapeutic Effects of Tuberculin.—Tuberculin as a therapeutic agent is based on results obtained in the laboratory through animal experimentation. It would be reasonable to exact that it should be efficacious in experimental tuberculosis in animals. But it is a fact that *there is no record in medical literature that any investigator has succeeded in curing or benefiting a tuberculous animal with tuberculin treatment.* In Robert Koch's writings at the time he introduced tuberculin we can find no clear-cut statement to the effect that he cured an animal with this agent. Klimmer, Lydia Rabinowitsch,[1] and others have recently tried small, very small doses, corresponding to those used at present in the treatment of human phthisis, but the tuberculous guinea-pigs and rabbits failed to improve. "No curative influence has been exercised by the tuberculin. The control animals lived sometimes longer than the treated animals. On the use of large doses the animals readily succumbed."

It has never been observed that the administration of tuberculin to tuberculous animals should promote healing of a tuberculous lesion, that cicatrization should be favored.

What has been observed, however, is that *very often dormant tuberculous processes are activated after the administration of tuberculin.* Bacilli which gave no trouble were released, "mobilized," producing a bacteremia, as was already mentioned (see p. 284).

Serologically, tuberculin has hardly ever shown its therapeutic value. Like other antigens, tuberculin stimulates the production of antibodies when inoculated into a tuberculous organism. But these antibodies cannot be considered true antituberculins because they do not neutralize tuberculin *in vitro.* We know that the antibodies produced by other toxins, as those of tetanus and diphtheria, neutralize

[1] Tr. Annual Conference Nat. Assn. Prevent. Consumption, London, 1913, p. 44.

the toxins of these infections *in vitro*, while the tuberculous antibodies do nothing of the kind. We can consequently see no theoretical or practical value in tuberculin from this viewpoint.

Clinical Evidence.—In a discussion on the merits of tuberculin treatment, Hector W. G. Mackenzie[1] said that "he should like to ask whether anyone has been able to obtain a cure of tuberculous ulcer, arising from the primary inoculation by means of tuberculin injections. He fears the answer must be in the negative."

We arrive at the same conclusion when we consider the clinical evidence presented by the advocates of tuberculin treatment in phthisis. All effective medication has its indications, contraindications, and limitations. True specific treatment is not free from these limitations, as is true of quinine, mercury, arsphenamine, thyroid, etc. But the limitations in the range of usefulness of these drugs depend mainly, if not entirely, on the presence or absence of mixed infection, of preexisting diseases, on the constitutional peculiarities of the patient, and complicating diseases. In a clear-cut case of syphilis in the average patient, arsphenamine or mercury will produce evident curative effects; malarial fever will be abated by quinine, myxedema is relieved by thyroid, etc. But in the purest forms of tuberculosis, in acute miliary tuberculosis, tuberculin is powerless, which fact alone should arouse suspicion as to its specific qualities.

It appears to be a general rule in pathology, as has been pointed out by von Hansemann,[2] that diseases which are not at times spontaneously cured cannot be cured by any known therapeutic measure. Rabies is usually mentioned as an exception, but even this may only be prevented; once it has developed, it cannot be cured. Specific therapeutics aims at curing diseases which are not known to be cured spontaneously. But it has never been observed that a patient suffering from acute miliary tuberculosis should be cured, the few cases mentioned by Cornet are all very doubtful. Acute miliary tuberculosis is the purest form of the disease without mixed infection; the tubercle bacilli, though disseminated all over the body, are found in each place in small numbers and they do not produce avascular masses from which medication is excluded. It should be the crucial test for specific treatment. As a matter of fact, however, tuberculin is altogether powerless in acute miliary tuberculosis, as it is in all progressive cases of phthisis.

Good results are reported by those who have used it in glandular, osseous, and articular tuberculosis in children. But we have already mentioned that these have a strong natural tendency to heal spontaneously in the vast majority of cases (see p. 471). Even surgeons advise and practise conservative treatment.

In phthisis the ideal cases are said to be those in the incipient stage of the disease. But when we recall that a really incipient case is one which has "slight or no constitutional symptoms, including particularly

[1] Tr. Annual Conference Nat. Assn. Prevent. Consumption, London, 1913, p. 9.
[2] Berl. klin. Wchnschr., 1911, **47**, 1.

gastric or intestinal disturbances or rapid loss of weight; slight or no elevation of temperature or acceleration of pulse at any time during the twenty-four hours," we are not surprised that many recover with tuberculin treatment. It was found that in Germany, France, and England many of those who were certified as tuberculous and eligible for sanatoriums, were fit for military service. Instead of sending them to institutions, as was the rule during times of peace, they were sent to the trenches and in the vast majority of cases they stood the hardships of war as well as other soldiers. Evidently many cases are abortive tuberculosis which under ordinary circumstances pass as chronic phthisis and any form of treatment gets credit for a cure. Tuberculin evidently gets its share of credit.

Lack of Reliable Statistics of the Efficacy of Tuberculin.—To prove its therapeutic efficacy, a specific must produce results in a larger proportion of cases of phthisis than is observed with the older methods of treatment. This has not been shown. In fact, there are no reliable statistics of large series of cases available. In their book on tuberculin treatment, Riviere and Morland state that they decided to give no statistics of results of tuberculin treatment, because they consider figures of questionable value. Sahli also gives no statistics, while the figures compiled by Brown in Klebs's book show clearly that there was no difference in results between the group treated with, as compared with those treated without tuberculin. Reliable statistics of ultimate result are not available at all.

Dosage.—It would be pretty bad for physicians, and for patients, if there was such a disagreement as to the dose of any potent remedy, especially if it was not known which quantity of the remedy is likely to be harmful. The initial dose ranges between 1 mg., recommended by Bandelier and Röpke, to 0.0000005 mg., recommended by Philippi. Between these two extremes, various authors recommend intermediate quantities, each one claiming that his standard is best, or, what is of more importance, the safest. Still, with such uncertainty as to dosage, many authors make tables of dosage and iron-clad rules as to gradual increase in the dose, and the final dose, some using logarithmic tables for their calculations, as if they were dealing with an exact science.

The fact is that there is no mystery about the technic of administration of tuberculin, and no knowledge of higher mathematics is necessary to make the various dilutions properly. Many pharmaceutical houses sell tuberculin in proper dilutions ready for use. But those who want to make their own dilutions can do it easily.

All that is necessary is six or ten amber-colored bottles of 10 or 20 cc capacity each. They are to be clean and properly sterilized. A larger bottle containing the diluent (sterilized, or distilled water containing 0.8 per cent of sodium chloride and 0.5 per cent of carbolic acid) should be at hand. Each of the small, colored bottles is to be filled with 9 cc of the diluent and marked with numbers, I, II, III, IV, V, VI, etc., respectively. Now take 1 cc of tuberculin and drop it into bottle No. I and shake it well. It now contains a 10 per cent

solution of tuberculin, so that a syringeful, with a capacity of 1 cc, contains 0.1 cc of tuberculin, or 100 c.mm.

When we take 1 cc from bottle No. I and drop it into bottle No. II, we get a solution containing 1 per cent of tuberculin; one syringeful contains 10 c.mm. of tuberculin. Repeating the process, dropping 1 cc from bottle No. II into bottle No. III, the latter will contain a 1 to 1000 dilution; 1 cc equals 1 c.mm. of tuberculin; bottle No. IV, a 1 to 10,000 dilution; bottle No. V, a 1 to 100,000 dilution; and bottle No. VI, a 1 to 1,000,000 dilution, so that a syringeful will contain a dose of 0.001 c.mm. of tuberculin. These dilutions may be carried further and the dose, which should always be small, if administered at all, may be infinitesimally so.

If given for its psychic effects, which is in fact done at present by most who use this agent, it is advisable to have ten bottles and that the first injection should be made from bottle No. X. If the patient is impressed by the treatment, he will "react" at least with 0.3° to 0.5° F., which should satisfy anyone who is looking for a "mild reaction."

Moreover, there is no difficulty in administering properly a series of ascending doses of tuberculin, and no higher mathematics is necessary for its successful accomplishment. Taking the first injection as a unit, we may increase the next injection by one-fourth or one-half. Thus, supposing we have used at first the dilution in bottle No. X containing 0.0000001 c.mm. of tuberculin per cubic centimeter, we inject but one-third or one-half of the contents of the syringe. The reaction is not likely to be severe, and we may one or two days later increase it to one-half or two-thirds of the contents of the syringe. In this manner we may proceed until we reach bottle No. VI, when the injection of a syringeful will give a dose of 0.001 c.mm. It is not advisable to give larger doses if we want to make sure that the patient is not harmed. But if there is any reaction the injections should be stopped promptly.

Utility of Tuberculin Treatment.—It cannot, however, be denied that some good results have been obtained with tuberculin treatment. Whether they could not be obtained with other methods in those cases is another question. Thus, E. Rist[1] says: "For my part, I have never seen a patient doing well under tuberculin without remaining in doubt whether he would not have done as well without tuberculin. Nor have I met with cases where the influence of tuberculin was so strikingly favorable that I could feel justified in letting them abandon the classical treatment and rely on tuberculin alone." Sir James K. Fowler says: "The tuberculin did not favorably influence the course of the disease in the majority of cases; in some cases the effects were detrimental; and even in stationary and improved cases it was difficult to ascribe any distinct improvement to the injections which might not have been equally attained under the treatment ordinarily employed

[1] Paris médical, 1913, **4**, 241.

in the Brompton Hospital." Likewise, Bardswell and Thompson,[1] in a recent statistical study of the experiences at Midhurst say that "collectively, the results point to the conclusion that tuberculin treatment, when given in addition to the usual measures practised in the sanatorium, had no appreciable effect either for good or ill. . . . Unquestionably many patients who received tuberculin treatment made good recoveries, but there are no grounds for supposing that they would not have progressed as satisfactorily had the injections been withheld: certainly, comparable cases treated without tuberculin gave no less favorable results."

In the extensive *Handbook on Tuberculosis*, A. Schröder[2] shows that "it has been established that in institutions for the treatment of tuberculosis in which only general treatment is applied, the lasting results obtained are not inferior to those reported from institutions in which, in addition to the general treatment, so-called specifics are administered."

Good results are obtained with tuberculin only when carefully administered in sanatoriums, with cases in the incipient stage, with but slight lesions, most of which are spontaneously curable. Although, according to Brown, at the Adirondack Cottage Sanatorium, no selection is exercised—the patients are allowed to elect tuberculin treatment. In private practice, as well as in most tuberculosis clinics in cities in this country, attempts with tuberculin have failed, evidently because the good surroundings, the fresh air, the proper food, regulation of rest and exercise were of more importance than the tuberculin. When we consider further that even the most ardent advocates of tuberculin state that only cases without fever, pursuing a slow course, showing no tendency to progress, but manifesting a strong tendency to fibrosis, are suitable for the treatment, it is clear that tuberculin is a remedy for those forms of phthisis which are spontaneously curable.

Psychic Effects.—We have seen that the tuberculous patient is very amenable to suggestion (see p. 294) and we have pointed out that in a certain class of cases tuberculin produces excellent results for this reason. On this point a large number of physicians agree, and they continue to administer tuberculin because of its psychic effects, although they may as well administer distilled water hypodermically and obtain the same results. To keep nervous, irritable, fretful patients for months, or even for years, is a difficult matter; often it is an impossible affair. Something must be done in addition to the rest, fresh air, milk, and eggs, of which he believes he knows as much as his doctor. Such patients, when given tuberculin, told to watch out for reactions, to record in detail the symptoms produced by each ascending or descending dose on a specially prepared blank, are often very much encouraged.

[1] Pulmonary Tuberculosis, Mortality After Sanatorium Treatment, Med. Research Com., Special Report Series, No. 33, London, 1919.

[2] Brauer, Schröder and Blumenfeld's Handbuch der Tuberkulose, 1915, **2**, 3.

This view of the psychic action of tuberculin is entertained by most authoritative physicians who use this agent extensively. Thus, Lawrason Brown,[1] who has done so much to popularize tuberculin in this country, says that only poor results can be expected when it is given "in cold blood." He believes "its value can be greatly enhanced when the administrator has implicit faith in its curative properties and imparts that faith to his patients." Another significant reason for using tuberculin treatment, according to Brown, "is the closer relationship that such treatment establishes between patient and physician. I must confess that I find it difficult to bring a patient to my office twice a week for months and discuss symptoms and fears, one of which gradually grows less while the other is often replaced by more or less indifference, born of familiarity. When, however, I give this patient tuberculin, he and I can discuss his case in detail twice a week and I am able to discover slight but important changes in his condition, to check imprudence, and to change needless timidity into confidence in his ability to order aright his life."

But similar results have been obtained by Mathieu and Dobrovici with "antiphymose," as was already detailed (see p. 657). In valvular heart disease, syphilis, myxedema, etc., this does not work.

I believe that I am safe in saying that, as a rule, tuberculin treatment is only efficacious in intelligent patients who are under the impression that they have mastered the theoretical aspects of infection and immunity and of specific therapy from reading popular books and articles on tuberculosis. In fact, in my experience, uneducated patients hardly ever improve under tuberculin treatment because they cannot understand the benefit of fever, malaise, pain in the limbs, nausea, debility, etc. On the other hand, intelligent patients look forward to the reaction as an indication that the tuberculin is "working on their system" and they often improve, provided infinitesimally small doses have been given.

There is no agreement among authorities as to what constitutes a "reaction" during tuberculin treatment. "All physicians are agreed that severe reactions are harmful to the patient, as a general rule," say Archer W. R. Cochrane and Cuthbert A. Sprawson,[2] "but there is still considerable difference of opinion between those who like their cases to progress without any reactions at all, and those who prefer mild reactions as a routine. Again, opinion varies as to what constitutes a mild reaction. In dealing with those otherwise running a normal temperature, the limit by some has been fixed at 100.4° F., and reactions thereto are disregarded; that is to say, these physicians will increase the next dose if the dose has not given a reaction over 100.4° F." But these authors consider this limit too high or dangerous, and are satisfied with a rise to 99.2° F. and call it a reaction. In other words, "the timid, or no-reaction school," treat only afebrile

[1] Am. Jour. Med. Sci., 1912, **144**, 524.

[2] A Guide to the Use of Tuberculin, London, 1915, p. 60.

cases. They should meet with immense success, because this class of patients recover spontaneously, or with any kind of treatment.

Dangers of Tuberculin Treatment.—Since the first use of tuberculin as a therapeutic agent, it has been recognized that it is capable of doing irreparable damage when imprudently administered. Virchow found that it produced rapid disintegration of the tuberculous tissues in the lungs, caseous pneumonia, and at times eruption of miliary tubercles. More recent investigations have shown that it often mobilizes the bacilli and thus may favor metastatic auto-infection. In fact, if phthisis was not a manifestation of immunity, disastrous results from this cause would be very frequent. It has also been observed that patients taking tuberculin for a long time are likely to develop nephritis. To be sure, with infinitesimally small doses the likelihood of such complications is reduced to a minimum, but the most experienced administrator is often surprised by unexpected reactions. I have seen such results repeatedly; mostly when tuberculin was administered by such as were not skilled in handling this potent agent, but also at times in patients who were treated by very skilful physicians.

Producing hyperemia of the affected lung area, tuberculin at times is effective in inducing pulmonary hemorrhage. When large doses were used this was very frequently observed and reported by Fränkel, Rumpf, Stricker, and many others. "Since small doses have been used," says J. Sorgo,[1] "with a view of avoiding strong reactions, hemoptysis is only rarely observed after the administration of tuberculin. At times small hemoptyses are seen, especially streaky sputum, but copious hemorrhages are rare. For this reason it is agreed that a tendency to hemoptysis is not altogether a contraindication to tuberculin treatment, provided strong reactions are avoided." But, as we already mentioned, this is not possible in every case. All who administer tuberculin for therapeutic purposes stop the treatment as soon as bleeding makes its appearance.

The general practitioner should not use tuberculin at all. He can obtain the same results by the judicious use of drugs without incurring any risk. Even psychotherapy of the kind applied by those who administer tuberculin can easily be practised with medication, as was shown in Chapter XXXIII.

Passive Immunization.—Active immunization through tuberculin having failed therapeutically, attempts have been made by many authors, notably Koch, Behring, Marmorek, Maragliano, and others, to cure the disease by passive immunization, or serotherapy. But the results have not been encouraging. It appears that so far we have not been able to raise the immunity to tuberculosis in animals to a degree that it may be transferred with the serum. Perhaps the reason is that, as far as our present knowledge goes, tuberculous immunity is hardly, if at all, humoral; in fact, it has been considered cellular by nearly all who have studied the subject.

[1] *Brauer, Schröder and Blumenfeld's Handbuch der Tuberkulose*, 1914, **2**, 255.

CHAPTER XLIII.

SYMPTOMATIC TREATMENT.

Cough.—To many patients the cough is the disease and they are under the impression that all they need for a speedy recovery is to be rid of this annoying and painful symptom. In its treatment some points are to be borne in mind: In most cases cough is decidedly conservative—a purposeful reflex act; it removes the secretions from the respiratory passages which, if retained, might act like foreign bodies or produce toxic effects. But, on the other hand, cough often disturbs the affected tissues which need rest, if cicatrization is to occur, or it may be responsible for insomnia, hemoptysis, pneumothorax, etc. Bearing in mind the importance of rest in the treatment of this disease, it is clear that a coughing patient is not at perfect rest, but hard at work. At times the reduction in the fever which absolute rest brings about, is obviated by the cough. Usually these conflicting principles can be reconciled by appropriate treatment.

Cough can be prevented or ameliorated by simple measures in a large proportion of cases. Atmospheric purity contributes considerably toward a reduction in its frequency and severity. Outdoor life and good ventilation of the room inhabited by the patient meet this indication. Mouth-breathing is a cause of excessive coughing in many cases, and some get fits of coughing when suddenly changing from a warm into a cold atmosphere, or the reverse. In steam-heated rooms, in which the air is usually dry, cough is more frequent than in rooms in which the air contains a proper amount of moisture.

In advanced cases with secreting cavities, the cough may be influenced by posture; reclining on one side, expectoration is facilitated, while lying on the other side brings about violent fits of coughing. Patients soon find out which position gives them relief and recline accordingly. However, it is best for some patients to recline on the side which induces cough and thus clears the chest securing rest in the interval. Many patients cough only during the morning hours and thus empty the cavities of the secretions which have accumulated during the night, while during the day there is but little cough. They need no treatment for this symptom.

It will be observed that some phthisical patients who sleep well during the night, cough more during the day than those who cough more or less during the night. The administration of large doses of opiates during the evening may gain relief in sleep, but also result in miserable hours during the following day. This is to be remembered when administering opiates to tuberculous patients.

Psychotherapy of Cough.—It is a noteworthy fact that the cough is greatly influenced by the psychic state of the patient. Persons with an irritable nervous system, the hysterical, emotional, and neurasthenic cough more than the dull, the phlegmatic and apathetic. Some cough while in the house, and are relieved as soon as they go out into the open air, while in others the cough increases as soon as the window is opened, or when they go out into the open air on a cold day. This last class of patients is very difficult to manage.

Other psychic influences are seen in patients who usually cough excessively, but cease when in agreeable company, or are intensely interested in something, etc. I have practically stopped unproductive cough in many patients by threatening them with expulsion from the hospital if they did not cease annoying their fellow-sufferers in the ward. Lonesomeness, and also insomnia are often responsible for excessive cough and should be treated according to indications. In sanatoriums the influence of example is often very good: The patient sees others control their cough and attempts to do likewise, and is often surprised at his success.

The patients can, within certain limits, control their cough, as Galen pointed out more than seventeen centuries ago, and Dettweiler has shown that this symptom can be "disciplined." Even when the cough is productive of considerable quantities of sputum, the patient is to be instructed that he need not expel it all at once; that if he succeeds in suppressing it for some time, the accumulated sputum will later be brought out with little effort. During the morning hours patients often make strong efforts to clear their chests. But if they wait till after breakfast they find in many cases that the sputum comes up easily. "Cough induces cough," says Penzoldt,[1] and for this reason patients are to be warned against giving in to the first tickling of the throat. The great struggle will only be during the first two or three days. Meeting with success, patients become convinced of their own powers to suppress, or control this symptom.

But patients must be warned in this connection against swallowing their sputum—"spitting into their own stomachs." Controlling does not mean entirely suppressing expectoration, as women and some men are apt to do. The dangers of the habit are to be explained in detail to the patient.

I cannot agree with those who prohibit smoking to tuberculous patients indiscriminately. To be sure, those who are not accustomed to tobacco often cough when near a person who smokes. But many habitual smokers are greatly relieved by a cigar or a cigarette. Our advice should be in accordance with the experience of the individual patient.

Many home remedies are very often efficacious in relieving cough. Thus, equal parts of boiled milk and honey or glycerin, with or with-

[1] Handbuch der Therapie, 1910, 3, 249.

out a flavoring agent, may be of great use in stopping an annoying cough. An excellent remedy is the application of a small mustard leaf or blister over the seat of the lesion. It may be repeated from time to time. The fact that it works by psychic suggestion should not deter us from using it, so long as the patient gets relief.

Medicinal Treatment.—After all the cases in which the cough may be controlled, or made bearable, by simple methods are discounted, there remains a large number who must be given sedatives to control this symptom. In the incipient stage these remedies are only rarely called for, and then only for a short time. But in advanced cases the indications for sedatives become more and more urgent. As Penzoldt says, the more progressive the disease and the less the chances of ultimate recovery, the more the charity of morphine is to be dispensed to the unfortunate sufferer.

In my experience, many cases in the incipient and moderately advanced stages of the disease are immensely relieved by creosote and its derivatives. The method of administration is given elsewhere. In those in whom internal administration does not relieve the cough, we may try the effects of inhalation of creosote, menthol, eucalyptol, tincture of benzoin, etc. The following is as good as any that has been recommended:

℞—Creosoti,
Acidi carbolici,
Spt. chloroformi . . . . . āā ʒiv 15.0
M. S.—Ten to twenty drops in an inhaler, to be used for fifteen minutes at a time.

Failing with these simple remedies we must resort to anodynes in case the cough is frequent, violent, paroxysmal, or disturbs the patient's comfort or sleep. Of these, cannabis indicæ is the least harmful and should be given the first trial. The extract may be given in doses of ¼ grain in pill or tablet form several times a day. In spasmodic cough it may be combined with hyoscyamus or gelsemium. The following may be used to great advantage:

℞—Extracti cannabis indicæ . . . . . gr. vj 0.4
Extracti hyoscyami . . . . . gr. xij 0.8
M. ft. pil. No. xxiv.
S.—One pill four to six times a day.

℞—Extracti cannabis indicæ fl.,
Extracti gelsemii fl. . . . . āā ʒij 8.0
Syr. acaciæ . . . . . ʒj 30.0
Aquæ menthæ piper. . . . . ad ʒiv 120.0
M. S.—One teaspoonful four times a day.

In many cases nothing but opiates gives relief. But in incipient cases opium and its derivatives are to be avoided because they may have to be continued for long periods and, in hopeful cases, the danger of habit formation is not negligible. In addition, opium deranges the digestive functions, produces anorexia and constipation, slows the frequency

and the amplitude of the respiratory movements, and favors stagnation of the secretions in the respiratory passages. A dose of Dover's powder may be given in the evening now and then with a view of controlling the cough during the night, but to continue the administration of opium in any form for any length of time is dangerous.

Of the many opiates, codein, which is ten to twelve times less toxic than morphine, is to be preferred. It may be given in tablet form in doses of $\frac{1}{8}$ to $\frac{1}{4}$ grain, and in advanced cases in much larger doses several times a day; or it may be added to any other medication that is being administered. Thus I quite often add it to creosote medication:

℞—Guaiacolis carbonatis	ʒiiss	10.0
Strychninæ sulphatis	gr. j	0.06
Arsenici trioxidi	gr. j	0.06
Codeinæ phosphatis	gr. viij	0.5

M. ft. capsul. No. 50.
S.—One capsule three times a day after meals.

℞—Codeinæ sulphatis	gr. iv	0.3
Extracti cannabis indicæ	gr. vj	0.4
Extracti belladonnæ	gr. iij	0.2
Extracti glycyrrhizæ	gr. xij	0.8

M. ft. pilulæ No. xii.
S.—One pill at night.

In most cases in which sedatives must be given for a considerable time the dose must soon be increased because after a few weeks the effects on the cough are diminished. Instead of increasing the dose, we may do better by changing one for some other derivative of opium. Heroin may be given in doses of $\frac{1}{24}$ to $\frac{1}{8}$ grain according to indications. It does not constipate and when there is dyspnea it is the best palliative remedy. It is to be regretted that because of its habit-forming potentialities, some hesitate in using it, and strong efforts have of late been made to discourage its use in practice. I have had no cause to regret prescribing it for these reasons, though I have used it in daily practice for many years. In incipient or favorable cases the chances of inducing a habit are remote; I have not met with a case. In far advanced and hopeless cases there is no harm done. Why not make the last earthly days comfortable? Dionin is another of these preparations and, when insomnia is a troublesome feature, it is even better than the above. Not many cases of habituation to dionin have been reported, but it is more apt to cause constipation than codein or heroin. The two last-mentioned preparations do not interfere with the expectoration of sputum; some even maintain that they assist in its expulsion. Whenever feasible, these narcotics are not to be given after midnight in order to avoid headache and debility during the morning hours.

The emetic cough is a very difficult symptom to control in some cases. I have seen some in whom it was responsible for a bad turn in an otherwise favorable case. Rarely, no food can be retained. In

many cases it may be relieved by avoiding heavy meals—taking several small meals during the day. The patient should recline in bed immediately after meals and avoid any exertion, and even speaking. But at times we must resort to medication. Some have reported good results from several drops of chloroform well diluted, or from bromoform. I have had cases in which only cocain administered before meals was effective in retaining nourishment in the stomach. The following prescription of Albert Robin may have to be resorted to:

℞—Cocain hydrochloratis	. . . . .	gr. j	0.06
Codein sulphatis	. . . . .	gr. j	0.06
Aquæ chloroformi	. . . . .	ʒij	60.0
Aquæ	. . . . . ad	ʒiv	120.0

M. S.—Tablespoonful after meals.

Expectoration.—In the average case of phthisis expectoration is a salutary phenomenon, removing, as it does, foreign, often toxic, material from the respiratory passages. At times it becomes excessive and annoying, but it should never be suppressed. In some cases with extensive excavations the amount of sputum brought up may be controlled within limits by posture. We advise our patients to recline in certain positions which favor the expulsion of sputum and thus empty the cavities of their contents. Relief may thus be obtained for the rest of the day. In cases in which the sputum is fetid—rare in phthisis—antiseptic inhalations may be tried. Creosote, iodine, menthol, eucalyptol, turpentine, etc., may be inhaled through an inhaler or simply dropped in hot water and inhaled.

Very often patients complain that they feel heavy on the chest and that if they could only bring up sputum they are confident that they would be relieved. Many drugs have been used for this purpose, especially the so-called expectorant remedies. It seems that all that is usually attained is a disordered stomach.

It appears from recent pharmacological investigation that there are no drugs which, when given in small doses, will induce more abundant secretion into the respiratory passages, stimulate the cilia of the bronchial mucous membrane to bring out secretions, or render tenacious secretions more easily movable from the bronchial walls to which they adhere. J. L. Miller[1] found that ammonium carbonate and ammonium chloride, and the emetic group of expectorants, as apomorphine and ipecac, when given in sufficiently large doses to animals, increase the bronchial secretion. Ammonia salts per os, in moderate doses equivalent to 2 mg. in an adult man, do not increase bronchial secretions in the dog. Apomorphine and emetin, when given to dogs in doses considerably greater than the ordinary therapeutic dose for man, do not excite increased bronchial secretion.

It is therefore absurd to give nauseating potions of ammonium salts, senega, ipecac, apomorphine, etc. All we may succeed in doing is to

[1] Am. Jour. Med. Sci., 1914, **113**, 499.

disorder the stomach, but the secretion in the respiratory passages remains unaffected.

In fibroid phthisis it has been found empirically that potassium iodide, or iodides in other forms, facilitate, expectoration and give immense relief to suffering patients. The same is true of other chronic afebrile cases of pulmonary tuberculosis. I have been using iodides in this class of cases with benefit for many years.

Fever.—Fever is an indication of active, often progressive phthisis, unless due to some complication. Its continued presence proves conclusively that the disease is spreading, even if the physical signs remain unaltered. It is at times neglected or overlooked because, unlike fever in other diseases, the patient in spite of a temperature of over 100° F. may feel quite comfortable, have a good appetite, and even gain in weight. But the entire future of the patient may depend on the treatment of the fever; neglecting mild febrile attacks means an invitation for chronic prolonged fever with lessened chances of recovery.

During the initial stages of the disease fever demands rest in bed, not so much as a cure but as a preventive against the extension of the process in the lung. It is remarkable that in many cases the fever abates within a few days or a week only through an improvement in the hygienic conditions and the diet of the patient, and placing him in a light and well-ventilated room. It is unfortunate that very few patients are willing to submit to perfect rest at this stage, claiming that they are not sick.

There are many advanced cases of phthisis with quite extensive lesions in which there is a daily rise in the temperature of 1 to 1.5° F., but the patients feel quite well and are even able to pursue their vocations. They need no active treatment because they have become habituated to the subfebrile temperature which may be regarded as their normal condition. In them the tubercle bacilli have actually become saprophytic and give them no trouble. In this class of cases it is only necessary to take steps to reduce the temperature when the patient is clearly suffering as a result of it; when the fever produces symptoms such as anorexia, restlessness, irritability, insomnia, etc.; or when he is losing in weight. I have observed many cases in which fever was due to overfeeding, and a reduction in the quantity of food promptly brought the temperature down to normal.

A sudden rise in the temperature in the course of chronic phthisis may be due either to an extension of the lesion, a new pneumonic process in a hitherto unaffected part of the lung, or to some complication. The former demands rest in bed till the temperature comes down to normal; in the latter the indications are in accordance with the pathological conditions which present themselves.

Patients are apt to attribute an attack of fever to "indigestion," but in my experience acute gastritis is a rather infrequent cause of pyrexia in phthisis, though a dose of calomel at times relieves an evanescent febrile attack. But then we may suspect that the relation

was merely coincidental, a frequent source of error in therapeutics. More often fever lasting several days is due to some intercurrent mild or severe infection. In hospital practice there is seen at times an actual epidemic of obscure infections; most of the patients in a ward are attacked during a period of a couple of weeks. The treatment is rest in bed and some antipyretic, like antipyrin, quinine, aspirin, etc. Complicating pleurisy, with or without effusion, may be the cause of a rise in temperature. In some women premenstrual, or menstrual, fever demands rest in bed periodically for a few days. The instability of the temperature in phthisis, which has been discussed in a previous chapter, is responsible for many febrile attacks. Any physical or mental exertion, worry, grief and anxiety may raise the temperature several degrees. Prophylactic and curative action is indicated along these lines. In many patients who have become "thermometer fiends," getting excited after reading the thermometer, it is best to prohibit the using of this instrument of precision, and to keep the fever chart away from them. Very often improvement takes place soon after these simple measures are taken.

The fever accompanying active phthisis demands active treatment. The main aim should be to remove it, or to prevent its occurrence. If we fail in this, we fail in our efforts at relieving the patient. It may very often be prevented by putting a patient to bed at the very first indication of a tendency to hyperthermia from any cause. Indeed, the neglect of mild febrile attacks is very often responsible for prolonged and even fatal fever.

In high continuous fever perfect rest is indicated, preferably in the open air, or in a room with wide-open windows, as has already been detailed in Chapter XXXVII. The patient is to be treated as though he is suffering from an acute disease, like typhoid or pneumonia. It is often surprising to note the prompt improvement after a rest in bed for a few days. Patients with a temperature at a high level for several months are often difficult to manage. When accompanied, as it usually is, by progressive loss of appetite, weight, and strength, they become discouraged and rebel against the prolonged and strict confinement. In such cases, provided the temperature is below 101° F., the experiment may be made of permitting them to leave the bed and get out in the open, resting on a reclining chair for a few hours during the day. The best hours are before or around midday, when the temperature is usually at its lowest; but any other time may be chosen under the guidance of the thermometer. In hectic cases the temperature is usually at its lowest in the morning and the patient may be allowed to leave his bed at that time. I have seen many patients, who did badly for weeks, improve when allowed to remain in the upright or semi-upright position for several hours a day. But care and circumspection are to be exercised while applying this treatment.

Some patients may be sent to the country and the change is at times effective in reducing the temperature when everything else has failed.

But this is not available to patients who have not the means to leave, accompanied by an attendant. Many authorities state that a mountainous climate is to be preferred for this purpose, but in my experience any change may do just as well.

It is deplorable that public sanatoriums do not admit febrile cases. Great service could be rendered by removing the patient for several weeks, during the period of fever, to better surroundings, giving him an opportunity to rest without interference by well-meaning, but often ill-guided, relatives and friends. I have often felt that cases under my care could be saved if sanatoriums were managed along hospital lines, admitting patients during acute exacerbations in the places which are now filled with patients whose condition is such that they would do well in any healthy surroundings which can be obtained in the average home.

When the temperature is not much above 100° F., the diet of the patient need not be different from that given in the usual case of active tuberculosis. Patients running higher temperature should not be starved. A rich, but easily digestible diet is indicated. Because of the anorexia, and actual distaste for food which these patients manifest, as a rule, the food must be appetizingly prepared, and here again a good cook can do a great deal. In many cases, to avoid cachexia in prolonged fever, some of the stomachic bitters should be administered with a view of improving the appetite. Whenever possible, milk should be used as a beverage in addition to the usual food, and given between meals. Some patients need alcoholic beverages and they should get them, because at times they are the best agent to improve the appetite and promote digestion. Patients with hectic fever are often benefited by some whisky an hour before the expected chill.

Hydrotherapeutic measures have not been found satisfactory in the treatment of fever in phthisis. The use of ice, or of cold sponging, or bathing, although possibly of temporary benefit, is contraindicated in most cases because they are apt to depress the patient. The most that can be done is to give a warm or tepid bath once or twice a week for the purpose of cleansing the body, but care is to be taken not to subject him to overexertion while going and coming from the tub. The fact that hydrotherapeutic methods have been given up in nearly all sanatoriums is sufficient proof that they have not been beneficial; in fact, that they are harmful.

Artificial pneumothorax is an excellent radical measure against tuberculous fever in appropriate cases. This will be discussed in Chapter XLV.

Antipyretic Medication.—Antipyretic drugs should only exceptionally be used in phthisis. In the first place, tuberculous patients do not, as a rule, suffer from the pyrexia to the same extent as patients with typhoid fever, pneumonia, etc., and a reduction in the temperature does not necessarily give the relief which the patient anticipates. It is not the fever, excepting hyperpyrexia, which is dangerous, but the

activity of the tuberculous process, and so long as only the former is influenced, the patient is not materially benefited.

The action of antipyretic drugs is ephemeral and deceptive, often accompanied by profuse perspiration which is enervating; and by digestive disturbances. Large and frequently repeated doses are necessary for weeks in the usual case and their action on the heart, which is not salutary, often leads to collapse.

But when the fever is high and continuous, or accompanied by headache, backache, and debility, one of the coal-tar antipyretics may give comfort with or without reducing the temperature. Acetanilid is to be avoided for well-known reasons. Phenacetin acts too quickly and produces profuse sweating. Antipyrin, or better pyramidon, may be used in 5- to 10-grain doses, combined with caffeine. Patients may stand the fever without complaining much, but in septic cases they abhor the chills which are apt to occur before the onset of the pyrexia. The best treatment is to place the patient in bed a few hours before the appearance of the chill, cover him well, and given him a drink of hot lemonade, tea, or whisky and, in severe cases, a dose of pyramidon. The chill may not be prevented completely in this manner, but it is rendered bearable. On the whole, antipyretic medication is to be administered an hour or so before the highest temperature is expected, varying with each case. Quinine should be given, if at all, five to six hours before the maximum temperature is expected, while pyramidon, antipyrin, aspirin, etc., require but two or three hours. When the fever has declined medication should not be continued, otherwise collapse may occur.

The salicylates are often very good in these cases, especially in the chronic hectic fever of consumption. The old prescription of sodium salicylate and arsenous acid (sod. salicyl., 10; acid. arsenicosi, 0.01; ft. pil. no. 100; S., five to ten pills three times a day after meals) is very good. But I have found that 7 to 10 grains of aspirin and $\frac{1}{150}$ gr. of arsenic in capsule three times a day are better. It is less likely to disturb digestion. But in patients showing a tendency to hemoptysis the salicylates are to be avoided. Pyramidon is best for this class of patients.

An excellent remedy for fever in tuberculosis is guaiacol painted with a camel-hair brush on the skin in 7- to 15-drop doses and covered air-tight. The temperature drops sometimes within one hour. It is best to rub into the skin of the thorax a teaspoonful of a 10 per cent guaiacol-vaseline ointment two or three times a day. It must be mentioned that collapse has been observed in some cases after the application of guaiacol.

Nightsweats.—No other symptom of chronic phthisis is more discouraging and enervating than nightsweats and their relief is of immense importance. It seems that in the vast majority of cases they can be prevented without the use of medication, and many physicians state that with careful prophylaxis they have not used any drugs for this symptom for years.

Open-air treatment is the best preventive of nightsweats. Sleeping in a cool room with sufficient, but not excessive, coverings must be enjoined. It is also good to give the patient before retiring a glass of cold milk with three or four teaspoonfuls of cognac to prevent the rapid sinking of the pulse-rate. In some cases a roll with butter may serve the same purpose. Some patients may be relieved by noting the time of the beginning of the sweating, and waking them a few minutes before and giving them an ounce of whisky. For private patients an alarm clock may be used for the purpose. This method, recommended by William Porter,[1] should be tried in all obstinate cases.

In cases in which these simple measures do not succeed, the sulphate of atropin in doses of $\frac{1}{100}$ grain, given in tablet form about seven o'clock in the evening, may give complete relief. Agaricin is also good in doses of $\frac{1}{20}$ grain, but it acts more slowly and must be administered about six hours before the sweating is expected. It often produces gastro-intestinal disturbances, especially diarrhea, and should be combined with an opiate—Dover's powder in 3- to 5-grain doses. Camphoric acid, in 10- to 20-grain doses, may be tried in obstinate cases. It is to be remembered that no remedy retains its power over this symptom for a long time, and after one ceases to act, we may try another.

Friction of the skin with tepid water, vinegar, or alcohol and water, or a 3 per cent lysol solution, may give relief.

Hemoptysis.—The prophylaxis of hemoptysis cannot be considered a simple matter despite the fact that we speak so much about the predisposing and exciting factors of pulmonary hemorrhage. Patients with really initial hemorrhages nearly always consult us only after the accident has occurred. Overexertion, excitement, etc., as exciting causes of pulmonary hemorrhages, have recently been shown to have no etiological relation in the vast majority of cases. It appears that most hemorrhages, especially those which are copious and fatal, occur during the night, or when the patient has been at rest. S. Bang[2] has recently made a special study of this problem and found that among 2000 tuberculous patients in a sanatorium, the initial hemorrhages came on while the patients were lying in bed, or on a reclining chair, in 69 per cent of 354 cases; in 15 per cent while they were dressing, sitting up in bed or just lying down; and in only 6 per cent of cases while the patients were walking or working; and in 8 per cent while they were otherwise engaged. In only 2 of the total number were the patients climbing stairs, though he estimates that these 2000 patients must have climbed the stairs over a million times, and taken 10,000 warm baths, and 25,000 douches while at the sanatorium. These facts, which may be duplicated by observations of any physician with large experience, show conclusively that overexertion is but a negligible factor, if any at all, in hemoptysis.

[1] International Clinics, Sixteenth Series, 1906, **4**, 77.
[2] Ugeskrift for Laeger, 1916, **78**, 419.

It appears that in active and progressive cases pulmonary hemorrhage is often the accompaniment of acute exacerbations of the disease. In rare cases we meet with hemoptysis, or even with fatal hemorrhages, in an entirely afebrile patient. But in most instances, fever, tachycardia, etc., precede the onset of the bleeding by several days. Bang's statistics substantiate this observation. Many patients suffering from acute exacerbations, or from febrile complications, have attacks of hemoptysis; at times, profuse hemorrhages. The prophylaxis in these cases is thus clearly the prevention of the acute exacerbations, or the febrile complications, which are liable to produce stasis and congestion of the involved lung area. The smaller hemorrhages are usually the result of diapedesis, being of parenchymatous origin, and have nothing to do with the position of the body, nor with overexertion or excitement.

The copious pulmonary hemorrhages, due to erosion of a pulmonary bloodvessel, can hardly be foreseen nor prevented; they are due to the involvement of a bloodvessel in the tuberculous process, with softening of its wall, thus allowing the blood to escape before a thrombus has formed. In others, it is due to the rupture of an aneurysm of Rasmussen, as was already shown in the chapter on Pathology. To speak in these cases of prophylaxis is futile.

All patients with pulmonary tuberculosis are to be told in advance that there is less danger in blood-spitting than is generally believed. We would thus avoid the psychic depression which is so often an accompaniment of hemoptysis. Women may be told that in the average case of hemoptysis there is no more danger than in the loss of blood during the menstrual period.

Not all cases of hemoptysis require the same treatment; individualization is required here, just as in most other pathological conditions. The vast majority of hemorrhages are insignificant, and if we only quiet the patient by an assurance that there is little danger, the bleeding will cease sooner or later, and the underlying process in the lung pursues its course uninfluenced by the accident. This is true of streaky sputum, which often terrorizes a patient to the same extent as a copious hemorrhage. But when the blood brought up is bright red, even if only a few mouthfuls, the matter is to be taken more seriously, because these small hemorrhages are at times the precursors of repeated and copious, though rarely uncontrollable, hemorrhages. As a rule, the fatal hemorrhages are copious and uncontrollable from the very beginning.

The patient is put to bed, but not in the traditional prone position. The blood and sputum must be evacuated from the respiratory passages with ease, and this can only be done when the patient is in the semi-sitting position. In this manner nourishment and medication can be administered without unduly disturbing the patient, expectoration is facilitated, and in copious hemorrhages, atelectasis of the posterior parts of the lung is prevented; eating, the administration of

medicines, vomiting, and the toilet, are thus facilitated. The time-honored ice-bag applied to the chest is of no value at all, excepting to keep the patient busy and attentive while attempting to keep it in place.

I have thus treated during the past five years nearly all the cases of hemoptysis under my care and found that the bleeding ceased just as quickly as when I applied the rigid-rest treatment. The psychic effect has even been more salutary. The patients are not so frightened as when they are warned that the least motion of the body, any word uttered, may increase the bleeding. It is best to place the patient in the semi-upright position immediately after the bleeding begins, because, as has been pointed out by Bang, rising in bed from the recumbent to the sitting position involves contraction of the abdominal muscles. These are liable to press upon the vena cava as in straining at stools, and by reflex action from the splanchnic nerve, cause an increase in the bleeding. This is probably responsible for the experience that sitting up in bed causes an increase in the flow of blood. It may be averted by placing the patient from the start in the half-seated position.

The therapeutic indications to be met are: Prevention of excessive cough and expectoration; increasing the coagulability of the blood and immobilization of the bleeding lung.

Morphine.—To allay excitement, procure rest, and thus prevent excessive cough, there is no better remedy than a hypodermic injection of morphine. We must bear in mind that we are in the presence of a conflicting situation. On the one hand, we must see to it that the effused blood in the bronchial tree should be removed; on the other hand, the strong expiratory efforts necessary to accomplish the expulsion of the blood and clots are accompanied by an increase in the pressure in the pulmonary circulation and, with their removal, the thrombi which plug the bleeding vessel are dislodged, and thus renewed bleeding is likely to occur. Morphine meets but one of these indications: It depresses the cough center, diminishes the frequency and amplitude of the respiratory movements, and quiets the mental state of the patient. Some have even found that morphine increases the coagulability of the blood. But after all it has its dangers. When given to excess, as is often done, it depresses the respiratory center, paralyzes the sensibility of the bronchial mucous membrane, and thus interferes with the expulsion of the blood and clots. Aspiration pneumonia may thus result in cases in which it is more successful as a hemostatic than is desirable.

For this reason morphine is to be used with great care and circumspection. Finding the patient excited and in agony, we inject hypodermically $\frac{1}{4}$ grain of morphine for its general and local effects. If the bleeding does not stop within an hour, the morphine should not be repeated, but other means are to be taken to control the hemorrhage.

Salt.—An ancient remedy for copious pulmonary hemorrhage is the administration of table salt. Formerly it was thought that because

it acts as an emetic, and thus depresses the blood-pressure, it is of use in hemoptysis. But we now know that its modus operandi is different. Von den Velden[1] has proved that, in man, swallowing 5 to 15 grams of table salt increases the coagulability of the blood within five minutes. Within one hour the coagulability returns to its former intensity. Sodium bromide has nearly the same effect. For this reason the administration of 5 to 10 grams of table salt, or 3 grams of sodium bromide, three or four times a day, may prove of immense value in hemoptysis. In very nervous patients the bromide is to be preferred.

More recently salt has been administered intravenously in isotonic solution, as recommended by Hans Müller.[2] Ten to 50 cc of a 10 per cent solution of sodium chloride, sterilized and heated to the body temperature, are injected into the median basilic vein, great care being taken not to drop any of the solution into the subcutaneous tissue, which is likely to cause intense pain. I have tried this treatment but have not found it superior to other methods.

Tying the Extremities.—The coagulability of the blood is also increased by tying up the blood in the extremities. A constricting band, or a tourniquet, is tied around the arm and the hip; two or three of the extremities are tied up at a time. In order to avoid injury to the nerves a roller bandage, or any other soft pad, should be placed under the tourniquet over the path of the larger vessels. The bandage should not remain in place for more than two hours, otherwise muscular paralysis or necrosis of the skin may result. As a rule, one-half hour is sufficient. The bandage is to be loosened slowly, by degrees, for obvious reasons.

Artificial Pneumothorax.—In cases in which the above measures are of no avail, the induction of an artificial pneumothorax may be considered, provided it can be ascertained in which side of the chest the bleeding is going on. This point is discussed elsewhere in this book. But it should be stated that in very acute cases, in which the exsanguination is sharp and brisk, there is usually nothing to lose and, even when we are not sure, we are justified in inducing a pneumothorax in the pleura of the lung which is most likely the source of the bleeding, as shown by clinical indications. When the bleeding lung is collapsed, the bleeding stops immediately.

Medicinal Treatment.—It will be noted that we have left to the end the numerous drugs which have been used for the purpose of allaying pulmonary hemorrhage. The reason is that *we do not know of any drug which will stop hemorrhage in the lung.* It seems to me that the reputation of some drugs as pulmonary hemostatics has been acquired on the basis of the fact that the vast majority of hemorrhages stop spontaneously; anything will do and receive the credit. This appears to be the consensus of opinion of phthisiotherapeutists at present,

[1] Ztschr. f. exper. Pathol. u. Therapie, 1910, **7**, 290.
[2] Beitr. z. Klinik d. Tuberkulose, 1913, **28**, 1.

although no less an authority than Albert Robin[1] says that he feels constrained to protest vigorously against the allegation that medicinal agents are impotent, and are only given credit for their psychic effects. To be sure, he says, there are many cases of hemoptysis which stop spontaneously, with or without treatment; there are others which cannot be controlled by any treatment. But between these two extreme types there are many cases in which medicinal treatment has a decidedly beneficial influence.

Emetin.—In former times emetics were given in hemoptysis and excellent results were reported because, with the vomiting, the effused blood in the bronchi was also expelled, preventing asphyxiation and also because the nauseous feeling reduced the blood-pressure perceptibly. Following Trousseau's suggestion, large doses of ipecac were given for this purpose. But we now have in emetin an excellent substitute for the nauseous ipecac. It acts as a hemostatic when many other agents have failed. I have used it in ¾-grain doses, repeated three to five times a day, with satisfaction. The simplest way of administration in these cases is hypodermically. Either the tablets or the ampoules, which many pharmaceutical houses prepare, may be used for the purpose. It appears that emetin is useful in cases of hemorrhage when there is no fever. Patients with fever above 101° F. are hardly ever, if at all, benefited by this drug.

The Nitrites.—The nitrites have been found efficient in checking the bleeding from the lung. They are known to lower the blood-pressure and this may be the cause of their efficacy. Macht[2] found experimentally that the nitrites cause a constriction of the pulmonary vessels and at the same time they are efficient peripheral and splanchnic vasodilators. As usually given in 2 or 3 drops, amyl nitrite is often inefficient. I found that J. E. Squire's[3] suggestion to give 10 to 15 drops, dropped on a handkerchief which is placed before the patient's mouth and nose, is best. Immediately the face becomes red and congested and the hemorrhage stops. It may be repeated several times during the day. In more copious hemorrhages, where the nose becomes blocked up with blood and clots, it may be necessary to put from 30 to 50 minims on a piece of lint and hold it over the patient's mouth. It may have to be repeated and the only complaint heard from the patient is that it produces a feeling of nausea. C. Fochi[4] says that when administered as soon as the first traces of blood-spitting are seen, copious hemorrhages may be prevented. But this is open to question. Fatal hemoptysis only rarely begins with streaky sputum. It is copious from the start, as a rule.

In slow bleeding, nitroglycerine, given in small and frequently repeated doses, as recommended by Flick, is often of service. When

[1] Thérapeutique uselle de la tuberculose, Paris, 1912, p. 294.
[2] Jour. Am. Med. Assn., 1914, **62**, 524.
[3] Clinical Journal, 1909, **34**, 155.
[4] Gazetta degli Ospedali, 1908, **29**, 114.

administered in 2- to 4-drop doses of the 1 per cent alcoholic solution it produces the same effect as amyl nitrite, but slower and more lasting effects are observed. Tablets are not to be trusted because they are often inert, as has been shown by George B. Wallace and A. I. Ringer.[1] The 1 per cent solution, as represented by the pharmacopeial spirits, is the best form in which glonoin should be administered. The following formula may be prescribed:

℞—Spirit. glonoini	℥j	4.0
Aquæ aurantii flor.	℥j	30.0
Aquæ destil. . . . . ad	℥iv	120.0

M. S.—One teaspoonful three or four times a day.

Adrenalin.—During recent years adrenalin has been used quite extensively for hemoptysis. It has been stated that it works well in cases where it is likely that the hemorrhage is due to the erosion of a medium-sized vessel, and that in acute inflammatory conditions of the lung it is contraindicated. It increases the heart action and contracts the bloodvessels, especially of the intestines, kidneys, and spleen, and thus increases the blood-pressure. But Gerhardt says that the bloodvessels of the lung are but slightly contracted, while Frey found that in a bleeding lung in a rabbit the vessels dilated and the flow of blood was increased after the administration of adrenalin, and Macht[2] found experimentally that it causes a powerful constriction of the pulmonary artery. Moreover, according to von den Velden, the coagulability of the blood is increased 50 per cent after the subcutaneous administration of the remedy. Clinical experience with this drug has not convinced the writer of its efficacy in hemoptysis and it has therefore been discarded.

Ergot.—Ergot has been given in large doses (a teaspoonful of the tincture every three or four hours; ergotin hypodermically). But it has been conclusively shown that it increases the pressure in the lesser circulation, just what we want to avoid. In the writer's experience it has never been of any value; often decidedly harmful. The same may be said about digitalis.

Atropine.—Atropine administered hypodermically, in doses of $\frac{1}{80}$ grain every three or four hours, according to indications, has been of more service than ergot or digitalis. Still, in some cases the writer has observed an increase in the hemorrhage soon after its administration.

Gelatin.—With a view of increasing the coagulative power of the blood, gelatin has been recommended by Dastre and Floresco,[3] though there is evidence that the Chinese used it as a hemostatic as far back as the third century. Four to 6 ounces of a sterilized 3 per cent solution of gelatin are injected under the skin of the abdomen or thigh. Great care must be taken in preparing the solution, as well as while

[1] Jour. Am. Med. Assn., 1909, **53**, 1629.
[2] Jour. Pharmacol. and Exper. Therap., 1918, **3**, 243.
[3] Compt. rend de la Soc. de biol., 1896, **3**, 243.

injecting it, because severe cases of sepsis, even tetanus, have been reported. Altogether it is not a harmless procedure—it is painful, leaves painful infiltrations at the site of the injection, often provokes fever, and is followed by urticarial eruption. If gelatin is used at all it should be given by mouth. The patient may be given jelly made from calves' legs, etc., or gelatin may be mixed with milk; or a concentrated solution may be administered per rectum. On the whole, its efficacy in pulmonary hemorrhage is problematical.

Calcium Lactate, Acetate, Chloride, etc., are other time-honored remedies given with a view of increasing the coagulability of the blood in doses of 10 to 20 grains repeated four to six times a day. Their utility is doubtful; all that may be said about them is that they are painless and harmless. Intravenous injections of a 5 per cent calcium chloride solution has been found to stop hemorrhages. The writer has tried it in pulmonary hemorrhages, using first small doses; 5 cc of the 5 per cent solution, then in larger doses, up to 30 cc. But no encouraging results have been attained.

Camphor.—Several authors have recommended camphorated oil, administered hypodermically, in pulmonary hemorrhage. Lunde[1] reports that the hemorrhage stops immediately after the injection of 3 cc of 20 per cent camphorated oil. In the experience of the writer, it is not superior in its effects to emetin, but it should be used in obstinate cases.

Blood Serum.—The use of blood serum in hemophilia has suggested its application in hemoptysis with a view of increasing the coagulability of the blood. Horse serum may be used in doses of from 20 to 40 cc subcutaneously. Inasmuch as at present diphtheria antitoxin is everywhere available, it may be used. But manufacturing chemists now have on the market appropriate preparations. It should not be used several times at long intervals for fear of anaphylaxis. I have tried it several times and was not favorably impressed with it.

Thromboplastin and Euglobulin, which have been prepared according to A. F. Hess's method, and found efficacious when applied directly to bleeding surfaces, have been tried by George Mannheimer and Stanley L. Wang[2] in the treatment of pulmonary hemorrhage. It appears from the published cases that these preparations have no effect on the bleeding.

Venesection.—With a view of producing a rapid fall in the blood-pressure, venesection has been used in desperate cases of pulmonary hemorrhage. In the days of indiscriminate bleeding, this was one of the standard therapeutic measures,[3] but even at present many

[1] Norsk Magazin for Laegevidenskaben, 1918, **79**, 1253.

[2] Am. Rev. Tuberc., 1917, **1**, 469.

[3] According to Sidney Colvin (John Keats, London, 1917, p. 384), John Keats, the youthful but consumptive English poet, was bled when he was frightened one night by the expectoration of blood. Keats stated that he could not be deceived in the color, which indicated to him that it was arterial blood, and that it was surely his death-warrant. He, however, lived for about twelve months after that pulmonary hemorrhage.

authors recommend it. Bonney recommends it when the blood-pressure is abnormally high, even in small initial hemoptysis, and also in bronchopneumonia following pulmonary hemorrhage, when the right heart is dilated and there are pulmonary edema, cyanosis and coma. More recently A. G. Shortle[1] urged this method again in cases in which the bleeding is seriously interfering with the functions of respiration. "The prompt relief to the impaired respiration is not the only benefit rendered in such cases. The coughing and struggling for breath, with the coincident inspiring of blood and sputum into the air cells is also stopped, and the development of bronchopneumonia may be prevented." In persisting hemorrhages it is also indicated, according to Shortle: "It is safer to bleed from the arm than from the lung."

Of course, this is rather heroic treatment, and involves great responsibility, especially when attending to patients in their homes. But in desperate cases, in which there is evidently nothing to lose, it may be given a trial when everything else has failed.

Diet in Hemoptysis.—In cases of slight hemoptysis with streaky sputum, or when a few mouthfuls of blood are brought up, the diet need not be changed. But in active and profuse hemorrhage all solid and hot foods are to be interdicted. Inasmuch as the first indication is to reduce the blood-pressure, we must restrict the quantity of fluids ingested. Sudden or rapid filling of the bloodvessels with water increases the blood-pressure and may lead to an increase in the bleeding. In European resorts, where phthisis is treated with mineral waters, hemorrhagic cases have been excluded ostensibly for the reason that excessive ingestion of water induces hemorrhage. In very copious hemorrhages, fluids should be given only for the purpose of allaying thirst—a couple of ounces at a time. Swallowing small pieces of ice serves this purpose best. Alcohol, coffee, tea, etc., should be discarded. Milk, eggs, scraped beef, etc., may be given in small quantities at a time.

Twenty-four hours after the cessation of the bleeding, irrespective of the clots expectorated with the sputum, we may begin to feed the patient guardedly. The general condition of the patient, as well as the concomitant symptoms, should be our guides. A cup of milk every hour or two, cream, a raw egg, and some scraped beef may be given. On the third day ordinary feeding may be resumed, so that about five or six days after the hemorrhage a standard dietary is reached.

Convalescence.—During convalescence, if there is no fever, or there are no other complications, the patient may be permitted to sit up in bed, or on a comfortable chair twenty-four hours after the cessation of active bleeding. The expectoration of clots, which continues for several days, as a rule, should not deter us from allowing the patient to sit up. Forty-eight hours after the stoppage of active bleeding I permit my

[1] Tr. Nat. Assn. Study and Prevent. of Tuberc., 1915, **11**, 147.

patients to walk around the room. I have not met with a case in which walking induced a new attack of hemorrhage. On the other hand, the resumption of exercises should be delayed, especially after profuse hemorrhages. The patient is more or less exsanguinated and weak. He needs rest and good nourishment to recoup. It is best that for two or three weeks after such a hemorrhage the patient should keep at comparative rest. The cough should be carefully controlled during that period and exposure, especially to intense sun rays, avoided.

Dyspnea.—We have seen that subjective dyspnea is rare in uncomplicated chronic phthisis, and that the patients are only rarely short-winded, if at all. In some cases this symptom demands treatment.

Toxic dyspnea, due to progressive disease of the lung, is best treated by rest. It is always accompanied by fever, and the treatment directed to remove the pyrexia usually helps along in the direction of relieving the air hunger. During acute exacerbations in the course of chronic phthisis, toxic dyspnea is very frequent and the treatment is clearly defined.

Dyspnea is often due to some preëxisting disease. This is the case with pulmonary emphysema, asthma, cardiac and renal disease. The treatment is that of the underlying pathological condition. In those having emphysema, or asthma, the iodides are very often of immense help, provided there is no tendency to hemoptysis. For the nocturnal attacks of dyspnea, morphine or heroin may have to be given.

Dyspnea may be due to some acute or subacute complication, such as pleurisy, with or without effusion, spontaneous pneumothorax, etc. The treatment is considered in the sections dealing with these complications. In the terminal stages of the disease the air hunger may only be relieved by large doses of morphine or heroin, and no patient should be denied these solacing remedies. The danger of habit formation should not be thought of at this stage of the disease. The severe dyspnea and cyanosis is here due to weakness of the atrophied respiratory muscles, limitation of the lung area owing to extensive destruction of the parenchyma, and, most commonly, to miliary invasion of both lungs with tubercles. The prognosis is hopeless.

Cardiac Weakness.—Patients who suffer from tachycardia or cardiac palpitation, permanent or provoked by mild exertion or excitement, must be kept at perfect rest in bed, and all forms of nervous and emotional excitement are to be avoided. At times these cardiac disturbances are due to gastric derangement and may call for modifications in the quantity and quality of the food.

In many cases, especially in the advanced stages, palpitation is due to cardiac displacement, especially in left-sided lesions in which the heart is drawn upward and to the left. Rest is the only remedy we have for this condition.

From whatever cause cardiac weakness arises, it may at times

become acute; collapse is not uncommon after some excitement or overexertion. Now and then a patient dies suddenly as a result of heart failure. For collapse, hot drinks of whisky, warm applications to the extremities, and some stimulants like camphor, strychnine, etc., are to be administered hypodermically.

In the far-advanced stages there is acute dyspnea, cyanosis, and edema, owing to cardiac failure resulting from the extensive lesion, toxemia, etc. These terminal symptoms are treated with digitalis, though in my experience this drug has only exceptionally an influence on the heart at this stage. In most cases the subjective feeling of weakness and air hunger are best relieved by liberal doses of morphine or heroin.

Insomnia.—In phthisical patients insomnia may be due to various causes, and it is not advisable to resort to soporific medication in every case. Rest and fresh air in the sleeping room may induce sleep; so may avoidance of a heavy meal late in the evening, a warm bath before retiring, etc. These means will suffice in most of incipient cases in which the sleeplessness is due to worry on account of the seriousness of the ailment. In some of these cases the bromides are very useful.

In incipient cases insomnia may be due to the cough which keeps the patient awake, and the indications are those discussed when speaking of the treatment of cough. When due to digestive disturbances, it is to be treated accordingly. In the advanced stages it is often due to the fact that the patient is lying at perfect rest during the whole day, and sleeps several hours, for an hour or so at a time. The patient is then to be kept awake during the day. In some cases hypnotic drugs must be given, and of these sulfonal or trional, in 10- to 15-grain doses, may be administered; 3 to 6 grains of veronal will serve the purpose in some cases. If the treatment has to be prolonged, the drugs may have to be alternated. In the far advanced stages only large doses of morphine may give relief.

Pains in the Chest.—Most of the pains in the chest complained of by tuberculous patients may be relieved by the administration of some placebo, or the application of a mustard plaster, dry cupping, tincture of iodine, etc. In some cases it is necessary to administer some of the coal-tar analgesics or salicylates. Small doses of antipyrin, phenacetin, pyramidon, etc., with caffein may be given. Sodium salicylate or aspirin gives relief in many cases. But in rare instances we meet with patients in whom the pains in the chest are so severe as to require the administration of a dose of codein or morphine. When due to intercurrent pleurisy, strapping of the chest with adhesive plaster is indicated. The pains in the shoulder, often due to diaphragmatic pleurisy, which are very acutely felt especially during the night, are very difficult to manage. The coal-tar analgesics and the salicylates usually give no relief, and often even safe doses of morphine fail. Hot

applications to the affected part, or, rarely, the actual cautery, may be necessary.

Anorexia.—Many patients have a good appetite; even when the fever is comparatively high the desire for food may be retained, which is not observed in other febrile diseases. But in others it is defective or inadequate to induce them to ingest a sufficient quantity of food for the replenishment of the inroads on their bodies made by the disease. It has been my experience that their number is not very large among those who are well instructed along the line of proper food and nourishment.

Medicinal treatment is not the first thing to give in anorexia. Outdoor life, regulated exercises, regularity of meals, etc., suffice in most cases to improve the appetite to the desired degree. In many it will be found that dietetic errors are at the bottom. The traditional and stereotyped advice, "plenty of milk and eggs," given indiscriminately, is more responsible for disgust for food than any other single factor. Drinking two or even three quarts of milk a day, and swallowing six to twelve raw or soft-boiled eggs, overload and often dilate the stomach, produce congestion of the liver, and create a disgust for all kinds of food. While some patients, who may be considered dietetic curiosities, may keep up with such a régime for weeks and even gain in weight, in the vast majority the digestive organs revolt, the palate loses its taste for food altogether and, coupled with diarrhea or constipation, the functions of assimilation fail.

In this class of patients we may note with satisfaction a remarkable change soon after the quantity of milk and eggs is reduced, or they are altogether discarded for a time. We must never neglect to tell our patients that so long as the appetite and digestion are good, they need not make any changes in their accustomed diet, excepting perhaps to increase the quantity, which is very desirable. With a variety of foodstuffs it is usually easy to consume more than before the onset of the disease. Instructions along the lines of good cooking should never be neglected. Among the poor and moderately well-to-do it has been my habit to send for the mother, wife or sister of the patient and urge her to exercise special care in the preparation of the food and to cater to the palate of the patient. The person who has prepared food for the patient for a long time knows best what he will relish. Of course, the teeth are to be examined and repaired in case caries is found, and proper instructions as to mastication are to be given.

In most cases the appetite can be improved by corrections of any of the just-mentioned errors without any medication at all. All are to be told in plain language that their only chance for recovery lies in consuming proper food and plenty of it; that they can best be cured through their stomach, and that they must eat even if the desire for food is not at its best. This often has the desired effect. When the patient finds that with proper food he gains in weight he is encouraged

to eat more. The gain in weight is usually seen best during the first month or two, but after a considerable increase the gain slackens. So long as he holds his own at his former weight, or little above, there is nothing to worry about.

Very frequently superalimentation is the cause of anorexia. In these cases it is advisable to try C. V. Spivak's[1] suggestion: The patient who lacks an appetite is told to omit one, two or more meals until the appetite naturally returns. Natural hunger, thus induced, at times improves the appetite and relish for food much better than any dietetic or medicinal procedure.

Gastric Disturbances.—In some cases we must resort to medication to provoke an appetite. I consider creosote as the drug which acts the best. Small or moderate doses of creosote or any of its derivatives —creosote carbonate, guaiacol, guaiacol carbonate, etc.—may be given and the appetite and digestion promptly improve. In others we may give bitter tonics—the tinctures of nux vomica, condurango, cinchona, etc. Orexin tannate is also good in 5-grain doses in powder or tablet form taken half an hour before meals. When there is diarrhea this drug is very good. I have used the following with good results:

℞—Tinct. nucis vomicæ		℥ij	8.0
Acid. nitrohydrochlorici dilut.		℥iij	12.0
Tinct. gentianæ comp.		℥ij	64.0
Tinct. cardamomi comp.	q. s. ad	℥iv	120.0

M. S.—One teaspoonful well diluted in water three times a day before meals.

The nux vomica may be replaced by condurango, and the nitrohydrochloric acid omitted, in cases in which they are contraindicated. In obstinate cases stomachic medicaments are to be changed often.

In hyperacidity dietetic changes are to be made according to indications, and it is always to be borne in mind that it may be due to overfeeding. Often medication is necessary. I have had good results with the following:

℞—Magnesii oxidi		℥iv	16.0
Sodii bicarbonatis		℥j	32.0
Extracti belladonnæ		gr. ij	0.13

M. ft chart. No. xxiv div.

S.—One powder three times a day after meals.

Or the following effervescent powder may be given: 30 grains of bicarbonate of sodium in one powder, and 10 grains of tartaric acid in another. Each of these is to be dissolved in half a tumbler of water, then added one to the other and swallowed during effervescence. Some are relieved by a tablet of $\frac{1}{100}$ grain of atropine sulphate given after meals.

[1] Colorado Medicine, 1918, **15**, 90.

Constipation.—Constipation is another of the troubles of the phthisical which often interferes with the favorable progress of the case. It is best combated by proper dietetic measures, especially increasing the quantity of fruits and vegetables, fresh and cooked. But mildly laxative drugs must be given in many cases. Before giving them we must make sure that it is not one of the anodyne drugs, codein, morphine, dionin, etc., which is responsible. Phenolphthalein appears to be the best, and 3 to 5 grains may be given, and next to it cascara sagrada in appropriate doses.

In the advanced stages, complicated by adhesive peritonitis, when diarrhea is apt to alternate with constipation, laxative drugs are to be used with caution. They may induce uncontrollable diarrhea. It is always better to first try proper changes in the diet, or the effects of some special food. Thus, I find that buttermilk will cause a movement of the bowels better than any medication in some tuberculous patients.

Diarrhea.—We have seen that diarrhea in the tuberculous is not always due to ulcerations in the intestines and that the latter may exist while the patient is constipated. In many cases the diarrhea is due to chronic catarrh of the bowels induced by swallowed sputum, and the patient is to be warned against this very bad habit. In others it is due to consumption of large quantities of raw milk, and particularly raw eggs, as has already been shown (see page 741), and this must be corrected.

In case the diarrhea is due to tuberculous ulceration or amyloid degeneration of the intestines, it is often very difficult to manage. The patient must remain in bed and appropriate changes be made in the diet. Fluids in general are to be reduced in quantity, especially cold drinks. The great majority of vegetables, salads, fruits—raw or cooked—pastries, rye bread, fats and sweets are to be avoided. While most patients tolerate milk very well, there are many who do not and, in obstinate cases, it is advisable to discard it for a few days and watch the effects. Bouillon and soups should be given without the addition of vegetables; eggs, butter, scraped or finely minced beef, boiled fish and oysters may be allowed, but no lobster. Of the vegetables and cereals allowed the following may be mentioned: rice, sago, etc., boiled in milk or served with cream, mashed potatoes, etc.

In many cases medicinal treatment must be given to control the frequent stools. The ancient "styptic" remedies, such as lead acetate, iron, alum, etc., are worthless in the vast majority of cases. But the modern preparations of tannin, such as tannigen, tannalbin, etc., are occasionally of service in large doses, and should be given a trial. The subnitrate of bismuth should be given in doses of 10 to 15 grains five or six times a day. But in most cases opium must be used, more or less. Bismuth or tannigen may be given in powders combined with fairly large doses of Dover's powder, or the official tincture of opium in 5- to 10-minim doses three or four times a day.

℞—Tannigeni	ʒiij	12.0
Bismuthi subnitratis	ʒvj	24.0
Resorcinolis	gr. ix	0.6

M. ft. cachet No. xviii.
S.—One cachet four times a day.

℞—Bismuthi subnitratis	℥j	32.0
Tinct. opii deodorati	ʒij	8.0
Aquæ cinnamoni . . . . . q. s. ad	℥iv	120.0

M. S.—One teaspoonful four times a day.

When bismuth subnitrate fails we may try the subgallate in 10- to 15-grain doses with or without opium. There are, however, many cases in which everything, even the administration of heroic doses of opium fails to stop the diarrhea, and we must be content with relieving the pains.

D. Mandl has had good results in rebellious diarrhea by the injection into a vein in the arm of 5 cc of a 5 per cent solution of calcium chloride. Saxtorph[1] reports encouraging results with this method and says that a large proportion of patients are freed from the symptoms of intestinal tuberculosis for quite a long time. A rather extensive experience with this mode of treatment has convinced the writer that only in a certain class of cases it has proved of value. When the diarrhea in a tuberculous patient is due to dietetic indiscretions, to catarrhal conditions of the mucous membrane, or to slight intestinal ulcerations, an intravenous injection of 5 cc of a 5 per cent solution of calcium chloride will give prompt and prolonged relief. In some cases it is necessary to repeat the injection once or twice at three-day intervals. In many cases the pains in the abdomen are relieved much earlier than the loose stools. On the other hand, when the abdominal pains and diarrhea are due to extensive ulcerations of the intestine, and amyloid changes in the mucous membrane, very little can be expected from this mode of treatment. Likewise, afebrile cases are more often benefited than those with fever.

Some of these patients complain of tenderness or pain in the abdomen. This is best relieved by hot fomentations. In the later stages, when emaciation is extreme, the extremities are to be kept warm and the unfortunate patient should not be denied the merciful relief of morphine in large doses.

[1] Ugeskrift for Laeger, 1918, **80**, 1763.

CHAPTER XLIV.

OPERATIVE TREATMENT—ARTIFICIAL PNEUMOTHORAX.

Historical Note.—Tuberculous pneumothorax has been the most dreaded of complications of phthisis and experience has taught that the vast majority of patients who suffer from this accident succumb. But some have observed that a pneumothorax may be what the French call "providential," and exert a rather salutary influence on the symptoms of the underlying disease. In a very interesting study of the physiology of respiration James Carson[1] suggested that the most rational treatment of phthisis would be collapse of the affected lung. So convinced was he of its possibilities, that he induced two patients to submit to the operation. The first was "James, Sloane Esq., an eminent merchant of Liverpool, the last of five brothers, the other four having died of consumption a few years before, who had returned from the West Indies, to which he had gone for the purpose of trying what a change of climate might do in his case, in the last stage of consumption, which he knew to be incurable from any known remedies. Soon after his return he heard of the paper to which I have alluded and soon became determined of having the operation which I suggested as a possible means of giving relief, performed in his own case. It was done on the 26th of September, 1822, by Mr. Bickersteth, an eminent surgeon of this place, in the presence of the late Dr. McCartney and myself. An incision calculated to admit air freely into the chest was made between the sixth and seventh rib. As the sound usually heard upon an opening being made into the chest, and produced no doubt by the rapid passage of the air through the opening was not perceived in this case, it was suspected that the lung did not collapse, and that an adhesion prevented the entrance of the air. It was not deemed advisable to make a further examination at this time." A second patient who submitted to a similar experimental operation also had pleural adhesions which prevented the entry of air into the pleural cavity. Carson's suggestion was forgotten for many years, though we find it mentioned in various books dealing with tuberculosis published during the first half of the nineteenth century. In his book on diseases of the chest, published in 1837, that acute clinical observer, William Stokes,[2]

[1] An Inquiry into the Causes of Respiration; of the Motion of the Blood; Animal Heat; Absorption; and Muscular Motion; with Practical Inferences, second edition, London, 1833.

[2] Treatise on Diseases of the Chest, New Sydenham edition, p. 455.

has this to say: "The proper symptoms of phthisis are in many cases arrested, and singularly modified, by the occurrence of the new disease (pneumothorax). I have often found that after the first violent symptoms had subsided, the hectic ceased, the phthisical expression disappeared, the flesh and strength returned; and in this way the patient has enjoyed many months of comfortable existence, and was only disturbed by dyspnea and the sound of fluctuation on exercise." In his book on *Diseases of the Lungs*, published in 1860, Walter Hayle Walshe[1] says: "In some recorded cases of actively advancing phthisis, the first sufferings of accidental perforation having passed, it has certainly appeared, though the signs of hydropneumothorax remained, that the phthisical symptoms themselves underwent improvement. But an occurrence so rare gives no warranty for the fanciful proposal to treat phthisis by producing artificial pneumothorax." This shows clearly that the method was suggested in England long before Forlanini had done it in Italy. During the course of the nineteenth century many other physicians have reported experiences similar to those of Stokes and Walshe just quoted.

It was, however, C. Forlanini,[2] of Pavia, who first induced a pneumothorax for therapeutic purposes, and reported his experiences in 1894. Independently of Forlanini, John B. Murphy,[3] of Chicago, did the same in 1898. But for some time no notice was paid to this method of treatment until Brauer, Spengler, and some others, took it up in Germany. At present it is one of the recognized methods of treatment of certain cases of pulmonary tuberculosis. *That it is a valuable method will be appreciated when it is borne in mind that it is mostly indicated in cases in which everything else has been tried and found wanting;* in other words, when there is everything to gain and nothing to lose. Contrasted with other methods of treatment, which are nearly always stated to exercise their alleged curative effects only during the incipient stage of the disease, when diagnosis is often doubtful, and spontaneous cures are not uncommon, it is to be considered one of the best therapeutic procedures we have at present for the cure of phthisis.

Principles Underlying the Treatment.—The aim is to introduce into the pleural cavity a sterile and harmless material which will collapse the lung in the affected, or more affected, side of the chest. The lung is thus put at rest and given an opportunity to heal. We have already seen that functional rest is as important in phthisis as in other diseases. In surgical tuberculosis rest has been more effective as a curative agent than all other methods. Rest has also been used with beneficial

[1] Practical Treatise on Diseases of the Lungs, American edition, Philadelphia, 1860, p. 250.

[2] Gazz. d. osped., 1882, **3**, 537, 585, 601, etc.; Gazz. med. di Torino, 1894, **65**, 381, 401. For a complete summary of Forlanini's work on this subject, see Forlanini, Die Behandlung der Lungenschwindsucht mit dem künstlichen Pneumothorax, Ergebn. d. inneren Medizin u. Kinderheilkunde, 1912, **9**, 621–655.

[3] Jour. Am. Med. Assn., 1898, **21**, 151, 208, 281, 341.

results in other diseases, notably general rest in functional nervous diseases, as was worked out by Weir Mitchell; tracheotomy in certain laryngeal conditions, gastro-enterostomy in cancer, and especially in ulcer of the stomach, enterostomy in certain diseases of the lower bowels and rectum, etc.

The lung is one of the organs of the body which never rests but expands and contracts at least 12,000 times per day throughout life. With an artificial pneumothorax we can place one lung at rest almost as effectively as the splint puts at rest a tuberculous joint, without endangering the life of the patient. Moreover, the lung is the only organ in the body which is constantly in a state of distention. Even after the most forced expiration it does not collapse utterly. Any solution in continuity in the pulmonary tissues remains separated and there appears to be no tendency to bring about the union of the diseased parts, or to facilitate the process of healing, by coaptation. Inflating gas into the pleural cavity and collapsing the lung, we achieve two objects: The lung is immobilized at its root, and it is compressed by the gas in the pleural cavity and the retraction of its elastic tissues. Its volume is greatly reduced, diseased parts and walls of cavities are brought into apposition, so that they may cicatrize by the formation of connective tissue.

Pneumothorax does even more than afford rest to the diseased lung. By compression it empties the lung of its contents. The pus and cheesy detritus in cavities, the inflammatory exudates in the alveoli and bronchioles are all squeezed out as from a sponge, removing the main source of toxic absorption. It also limits the diseased focus and prevents its spread, so that the healthy parts of the lung remain so while the lesion is in time converted into a cicatrix, or is encapsulated. As a result of drainage, mixed infection is eliminated and prevented. The fact that the air current entering through the trachea cannot circulate within the collapsed lung tissues prevents superinfection of healthy parts of the organ with emboli of detritus carried from one part to another along the bronchial tree, and mixed infection with micro-organisms other than tubercle bacilli, which may be brought in with the air current, is avoided.

The circulation of the blood is impeded in the collapsed lung, but there occurs a venous or passive hyperemia which is known as an important factor in the defence of tissues against tubercle bacilli. The comparative protection against tuberculosis enjoyed by cardiacs is ascribed by some authors to the venous hyperemia of the lungs. The lymph channels of the collapsed lung are compressed, as has been shown by Shingu,[1] who subjected animals with induced pneumothorax to the inhalation of soot, and at the autopsy found that the collapsed lung remained free from soot. Animals were compelled to inhale large quantities of soot, and subsequently pneumothorax was induced,

[1] Beitr. z. Klinik. d. Tuberkulose, 1908, **11**, 1.

and when they were finally killed it was found that the free lung was darker than the collapsed lung. This tends to show that the circulation of lymph, which is the main factor in removing inhaled particles from the lung, is impeded or arrested because of stasis of lymph in the compressed lung. In this manner the absorption of toxins from the lesions into the general circulation is impeded or arrested in pneumothorax, the clinical phenomena of phthisis, such as fever, nightsweats, weakness, etc., are prevented, and the body is thus given an opportunity to recuperate. Moreover, the lymph stream being unable to carry away bacilli from the lesion, the process is localized to the affected areas. These points have been found clinically, at the autopsy table, and experimentally.

Technic.—The technic of the induction of a pneumothorax is simple, but not devoid of danger and even fatal accident. The object is to inject gas into the pleural cavity and not anywhere else. Forlanini developed a technic which is both painless and bloodless. Murphy, without knowledge of Forlanini's work, developed a practically similar technic. Brauer was not satisfied that the Forlanini-Murphy method is safe and advocated the open incision method.

The Brauer Method.—This consists in incising the chest wall, dissecting down to the pleura by cutting through the fascia, and separating the intercostal muscles with a blunt instrument in the direction of their fibers. When the parietal pleura is exposed, it is punctured with a blunt needle or cannula, and the gas is allowed to flow in by aspiration of the pleural cavity or by pressure, when indicated. This method has failed to get many adherents for many reasons. But few patients want to submit to a cutting operation. Then there is an obvious danger of sepsis which may, of course, be avoided by the usual methods. I have found no reason for resorting to the bloody operation, and feel confident that if this was the only available method of inducing an artificial pneumothorax we should find very few patients willing to submit.

Very few now practice this open incision method, and most of those who do it make use of it only occasionally when the Forlanini method fails because of pleural adhesions. It is however, a fact that when the Forlanini method fails, the open incision almost invariably fails to find a non-adherent pleural sac.

The Forlanini-Murphy Method.—It consists in a simple, bloodless puncture of the chest wall with an especially constructed hollow needle which is connected with a gas reservoir and a water manometer through a T-shaped tube. When the lumen of the needle punctures the costal pleura the gas is allowed to flow into the pleural cavity by the suction or negative pressure in that cavity, as well as by some positive pressure which must, at times, be used at the gas reservoir.

Simple as this operation appears to be, there are certain difficulties to be overcome and dangers to be avoided. The main difficulty is to pass the needle as far as the costal pleura, puncture it, and avoid pene-

trating the visceral pleura and the lung. The dangers are mainly in allowing the gas to flow into places other than the pleural cavity, especially into a bloodvessel, thus causing gas embolism, which while not invariably fatal, yet is sufficiently menacing to be dreaded by all who are doing this sort of operation.

No special preparation of the patient is indicated. Brauer and Spengler[1] do not perform it in women during the menstrual period; Saugman avoids even the premenstrual period.

FIG. 120.—Robinson's modification of the Brauer apparatus for inducing pneumothorax.

Apparatus.—To avoid accidental entry of gas into bloodvessels or any other tissue but the pleural cavity various forms of apparatus have been invented. As is usual, they are all based on one main principle—the manometer which was introduced by Saugman. Each apparatus consists primarily of two graduated bottles connected by tubing, one containing the gas to be injected, and the other some fluid, so that the fluid flows from its container into the other bottle, displacing the gas which is sucked or pressed into the pleural cavity through a tube and an especially constructed needle. This last-mentioned tube is T-shaped, or provided with a three-way stopcock, of

[1] Handb. d. Tuberkulose, 1919, **3**, 192.

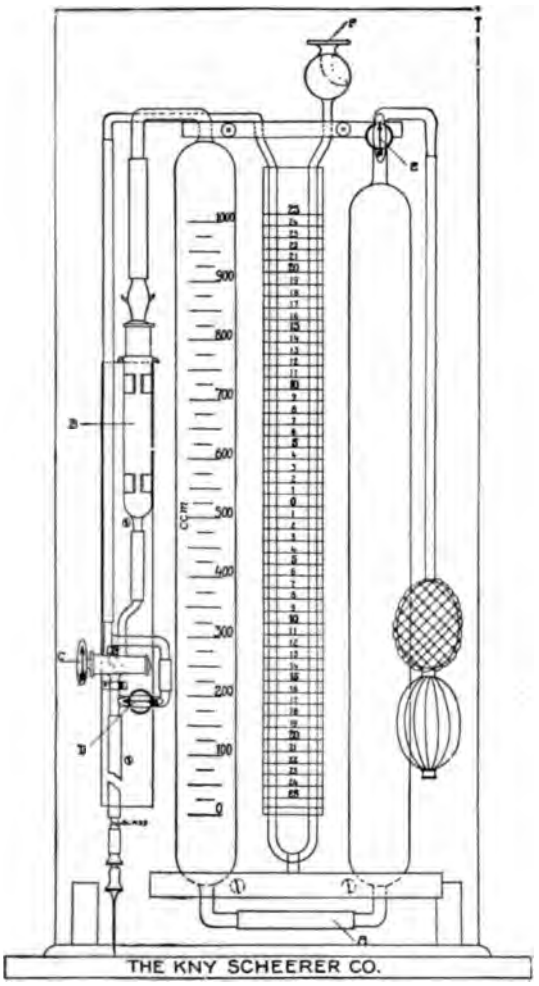

FIG. 121.—Forlanini-Saugman-Muralt apparatus for the induction of pneumothorax. This apparatus consists in the main of two glass tubes, twenty-four and a half inches high and about two inches in diameter and a U-shaped manometer tube, the latter filled with an alcoholic solution of methylene blue and mounted in the center of the board in front of a graduated porcelain scale. The two large tubes are joined by means of rubber tubing under the base *A*. The tube to the left is graduated to 1000 cc and the other is plain. They are filled with water up to 500 cc. The graduated tube to the left is filled from the tank with the gas to be introduced into the pleural cavity, and the gas displaces the water which rises correspondingly in the large plain tube to the right. When filling the apparatus with gas, the rubber tubing from the tank is to be connected with a rubber gas-bag to the opening below the stopcock *C*. Stopcock *D* should stand vertically. Stopcock *C* should be turned so as to connect through the filter and into the graduated cylinder. Stopcock *E* on the top of the non-graduated tube should be turned so as to allow the air in this tube to escape when the gas forces the water into it. When the graduated cylinder is full of gas, stopcock *C* should be closed. Funnel *F* connected with the manometer tube serves for the filling of the manometer tube to zero with an alcoholic solution of methylene blue. The graduated glass tube is connected with the glass tube *B* which is filled with sterilized gauze and serves as a filter. The three-way stopcock *C* connects with the manometer as well as the gas cylinder, thus showing the oscillations when the needle is in the pleural cavity. When stopcock *D* is turned horizontally it permits the manometric reading showing the degree of oscillation while the gas is still flowing. After the needle has been properly inserted into the pleural cavity and stopcock *C* turned to the graduated tube, the gas will be forced out by the weight of water which is contained in the plain tube. When extra pressure is required, a small rubber tube is connected with the plain tube, so that the remaining water may be gently forced into the graduated tube. The manometric scale is divided into 50 centimeters, 25 above and 25 below zero, indicating respectively negative and positive pressure.

which one limb communicates with the gas bottle, the second with the needle, and the third with the manometer. At any moment during the operation we can open or close the tube leading to the manometer or the gas reservoir.

As has been said, all the instruments for the induction of a pneumothorax are constructed on this simple principle, but it is amazing how some have succeeded in complicating them by adding various attachments which make them unwieldy, and easily disordered. The universal experience that a machine in order to be successful must be of the simplest construction consistent with efficiency, holds good here. I have been using Forlanini's apparatus as modified by Saugman[1] and von Muralt,[2] (Fig. 121) and also the Robinson apparatus (Fig. 120).

The Function of the Manometer.—The entire safety of the operation lies in the manometer which has been called by Edward von Adelung[3] the heart of the apparatus. While the needle passes through the skin, subcutaneous tissue, muscles, and fascia before piercing the costal pleura, the manometer records atmospheric pressure, but as soon as it enters the pleural cavity the air in the connecting tube becomes rarefied, because the vacuum in the pleural cavity aspirates its air content, and the fluid in the closed limb of the manometer is sucked up toward the needle, *i. e.*, from the open into the closed limb, and a distinct difference in the levels of the fluid is evident. Moreover, when the lumen of the needle is really in the pleural cavity, the respiratory movements of the lung are recorded in the manometer which shows distinct oscillations of the levels of its fluid.

This explanation of the work of the manometer, which is found in most works on the subject, is unsatisfactory. The fact is that normally there is no pleural cavity at all because the parietal and visceral pleura lie tightly, one on another; nor can we speak of negative pressure between the two pleural sheets because the word "pressure" is here used in the sense of gas pressure which can be measured with a manometer; but such a negative pressure does not exist between the two pleural sheets. The manometric readings, when the lumen of the needle is in the pleura, are better explained by Brauer, Piéry,[4] and Moritz,[5] and especially elaborated by Rist and Strohl,[6] in the following fashion: The lung must be considered as an organ fixed at its root, and kept in a state of equilibrium by the pressure of the atmospheric air within the air passages, and by the elastic tension of its tissues. There is a constant tension of the lung from the roots to the periphery at the thoracic walls. The force of this traction is equal to the absolute elastic tension in the given direction, minus the atmospheric pressure which prevails within the air passages and so prevents its collapse, or retraction, from

[1] Beitr. z. Klinik d. Tuberkulose, 1914, **31**, 571.
[2] Ibid., 1910, **18**, 359.
[3] Jour. Am. Med. Assn., 1914, **42**, 1914.
[4] La pratique du pneumothorax artificiel en phthisiothérapie, Paris, 1912.
[5] München. med. Wehnschr., 1914, **61**, 1321.
[6] Annales de Médecine, 1920, **8**, 233.

the periphery to the hilus. The intrapleural pressure, therefore, never differs much from the atmospheric pressure, as has been shown by W. Parry Morgan,[1] and in consequence any gas drawn into the cavity will not be appreciably rarefied. The volume of gas which will have passed from the connecting tube into the pleural cavity will be practically equal to the amount of fluid which will have passed from the open to the closed limb of the manometer. This volume would, when the negative pressure stands at 15 cm. of fluid in a manometer tube of 0.3 cm. bore, measure less than 1 cc.

This is enough to separate the sheets of the pleura, if there are no adhesions. But, owing to the elastic tension of the lung and the atmospheric pressure within the air passages, there is actually shown a negative pressure in the manometer. A little reflection will explain why this negative pressure will be stronger during inspiration because of the greater distance at that period between the root and the periphery,

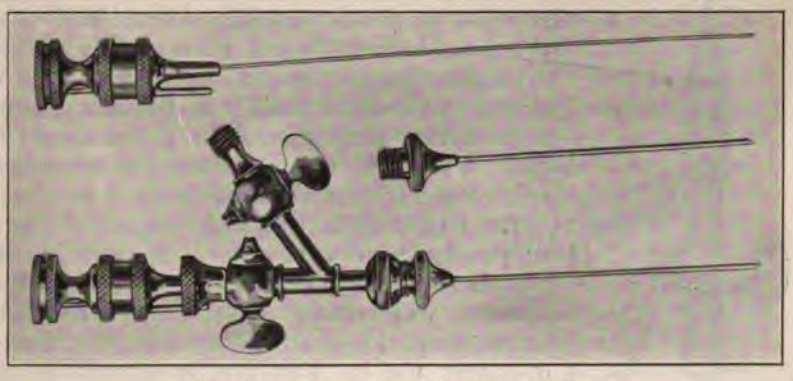

FIG. 122.—Brauer-Floyd-Robinson needle.

and less during expiration. With the increase in the quantity of gas introduced into the pleural cavity the tension of the lung will obviously decrease and with it the negative pressure, until finally a point is reached when the pressure in the gas-containing pleural cavity is 0 and later even becomes positive.

Bearing in mind these simple principles of the manometer, we are in a position to guard against the most important of the accidents which are liable to happen during the operation. In patients with pleural cavities free from adhesions, ordinary and careful attention to the manometer will suffice to guard against mishaps. The manometer shows conclusively whether the lumen of the needle is in the pleural cavity or not. It also gives reliable information as to the state of the pleural cavity with particular reference to adhesions, showing whether they are dense and extensive, or of slight extent and may be separated and broken up by an increase in the intrapleural pressure with the gas.

[1] Lancet, 1914, **2**, 90.

During the course of the treatment we are able to ascertain, with the aid of the manometer, whether the nitrogen has been absorbed and a refill is necessary; whether the lung has been completely immobilized or has remained expansile. When it is found that the intrapleural pressure increases, and this cannot be attributed to excessive gas insufflations, it indicates pleural effusion. The difficulties in cases with pleural adhesions will be discussed later on.

The Needle.—Various, some rather complicated, needles have been devised for this operation. The fact is that any trocar and cannula may serve the purpose; in fact, an ordinary hypodermic needle has been used successfully. For the first operation it is, however, best to use one with an obturator, which prevents the admission of air, an arm right below the obturator, to which the tube leading to the gas bottle and manometer is attached. As is stated elsewhere, the needle supplied is usually too long; one a little more than one inch in length is best. For the first operation the gauge may be over 1 mm., but for subsequent refills, especially in patients showing high suction of the pleura, the gauge should be from 0.4 to 0.8 mm. at most. Subcutaneous emphysema is often the result of thick needles.

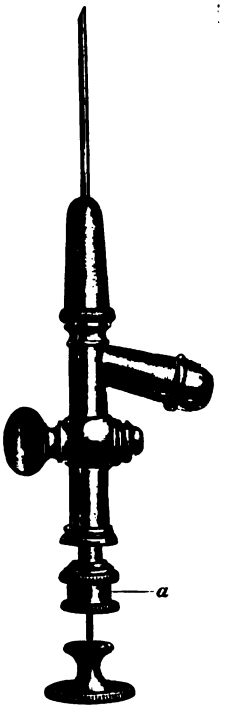

FIG. 123. — Saugman needle.

The Gas Used for Inflation.—Because it was supposed that when oxygen is injected into the pleural cavity it is quickly absorbed, and that nitrogen will remain within that cavity for a longer time, this element was selected and most operators use it. But further experience has shown that atmospheric air is just as good. Webb, Gilbert, James and Haven,[1] Tobiesen[2] and especially Tachau and Thilenius[3] and Rist and Strohl,[4] have shown clinically and experimentally that nitrogen has little if any advantage over atmospheric air, because in either case diffusion of gases occurs so rapidly that within a few hours the proportion of the two gases, nitrogen and oxygen, is about the same. For this reason there is no necessity for using nitrogen. Air does just as well. Nitrogen is rather expensive when bought in tanks from manufacturers, and while most of the apparatus for the production of pneumothorax is portable, the large iron tank of nitrogen is not easily transported, and atmospheric air is to be given preference in private practice.

[1] Arch. Int. Med., 1914, **14**, 883.
[2] Brauer's Beiträge, 1911, **21**, 109.
[3] Ztschr. f. klin. Med., 1916, **82**, 199 and 282.
[4] Annales de Médecine, 1920, **8**, 233.

The Selection of the Point for Injection.—The first inflation must be carefully done, and it is important to select a point to introduce the needle where no adhesions are likely to be encountered. Bearing in mind the anatomy of the chest and its viscera, it is evident that the ideal point is between the anterior and posterior axillary lines, in the fourth to the sixth intercostal spaces. Here pleural adhesions are comparatively uncommon, and in left-sided lesions the needle is not likely to plunge into the heart, a complication which has occurred twice in the hands of a very skilful operator. It must be mentioned that this accident proved harmless ultimately, though the immediate effects were threatening. When the patient lies on the opposite side and draws his arm above the shoulder, the intercostal spaces are widened, and the tissues hardened so that the needle passes easily through, obviating the use of force. But because at the mentioned region the interlobar fissure reaches the periphery, there are apt to be adhesions at the fourth or fifth intercostal spaces, and for this reason it is best to try first the seventh or eighth interspace, where the chances of entering the pleural cavity are better. When the lesion is mainly in the lower lobe, or there are unmistakable signs of pleural adhesions over the lower lobes, the puncture should be made in one of the upper, the third or fourth interspaces. Of course, when we are free to choose, areas covered with thick muscles, or the thick mammary gland in women, are to be avoided. But we are not always free to choose, and any point must serve our purpose when the elective places are not available because of adhesions. It must also be emphasized that it is very difficult, often impossible, to avoid pleural adhesions with all the means of diagnosis at present at our command.

We are generally guided by the following principles: The chest is punctured as far as possible away from the main pulmonary lesion because pleural adhesions are most likely to be encountered over the diseased lung and, what is more important, while puncturing the lung is ordinarily harmless, in such places the needle may, however, penetrate a cavity and produce a pyothorax. But adhesions are found everywhere, and often where we least expect them. Physical diagnosis is apt to prove misleading, and the fluoroscope and roentgenography just as often may fail to reveal them. I have met with cases in which the roentgenogram showed all the conventional signs of pleural adhesions but puncture revealed a free pleura, and complete collapse was easily obtained with three or four inflations. More often yet the roentgenogram shows a clear picture and it is concluded that the pleura is free, but puncture shows conclusively that there are adhesions. One sign of freedom from adhesions should be emphasized: I have invariably been able to introduce gas into a pleura over which friction sounds were audible during auscultation. On the other hand, feeble breath sounds, or complete absence of breath sounds, is in most cases an indication of adhesions.

Pleural adhesions are almost always present in cases with a history

of pleurisy with effusion in the recent or distant past. Indeed, if during the treatment an effusion occurs, its absorption is in most cases followed by adhesions, irrespective of the measures we may take to prevent their occurrence, and we cannot again induce a pneumothorax when indications arise. On the other hand a history of dry pleurisy is no indication of pleural adhesions. Many cases remain with free pleural cavities.

Forlanini is guided by tidal percussion of the margin of the lung, especially at the base. When he finds that the base line in the axilla shifts between 10 and 12 cm. during extreme inspiration, as compared with extreme expiration, he is convinced that the pleura is free. Good mobility of the lung margins is the most important sign of freedom from pleural adhesions, according to Forlanini, but he adds that immobility is not a sure sign of such adhesions and of obliteration of the pleural cavity. There are cases of extensive hepatization of the lung in which the mobility of the lung margin is defective or absent, yet the pleural cavity is free.

It appears that *the most reliable means of ascertaining whether or not the pleura is free is the attempt to enter it with the needle connected with a manometer.* In case the first puncture does not yield negative pressure in the manometer—a very frequent occurrence, so that when one enters successfully with the first puncture he considers himself lucky—another attempt is made at a different point. I have made in one case four punctures before succeeding in entering the pleural cavity and in another twelve before giving up the case as not suitable for the treatment. Forlanini made fifteen punctures in one case before he finally succeeded. It should be mentioned that at times failure to find negative pressure is no positive indication of complete obliteration of the pleural cavity by adhesions. In one case in which I could not enter after trying twice, a spontaneous pneumothorax occurred later. Giesemann[1] reports 3 cases, and Sedlmeyr[2] 1 case of spontaneous pneumothorax after failure to produce one artificially.

The skin at the site selected for puncture is painted with tincture of iodine and the excess is washed away with alcohol. It is then frozen with ethyl chloride and an injection of one-third of a grain of novocain or cocain in 1 to 2000 adrenalin solution is made. At first the skin is infiltrated, then a few drops are injected into the intercostal muscles, and finally into the pleura. The latter must not be neglected; it appears to be the only known way of preventing pleural shock, of which we shall speak later on.

Thoracocentesis.—The patient is always in the recumbent position during the operation, either on an operating table or, preferably, in his bed. With a view of widening the intercostal spaces, the hand of the side to be operated upon is placed over the head. The selected intercostal space is carefully palpated with the index and middle fingers of the left hand to make sure of avoiding a rib when thrusting the

[1] Beitr. z. Klin. d. Tuberk., 1918, **33**, 215.
[2] München. med. Wchnschr., 1921, **68**, 949.

needle into the chest wall. If a blunt needle is used, the skin is first punctured with a tenotome. The needle is inserted and pushed slowly forward, passing through the subcutaneous tissue, fascia, and muscles. While the latter are passed the needle goes smoothly, but when the endothoracic fascia is reached a certain amount of resistance is encountered, which is characteristic to the experienced hand. Often a snapping sound is audible. A similar but stronger resistance is felt when the pleura is passed and it is often difficult to decide with confidence as to whether it was the fascia or pleura which was punctured. "Never move the needle sidewise, for if it should be in the lung the latter may be easily torn by it." (Balboni.) The manometer is the only means at our command to make sure of where the lumen of the needle is.

How far the needle is to be pushed depends on the thickness of the chest wall of the given patient. All efforts are to be made to avoid penetrating the lung. While in the vast majority of cases this is entirely harmless, in rare instances it may prove a serious, and even a fatal accident. We may induce a spontaneous pneumothorax, an accident which occurs more often than is generally appreciated.

The usual length of the needle, Floyd's modification of Brauer's, is 5 to 6 cm. This is excessive and Saugman's needle, which is only 3 cm. long, is at present used by me exclusively. Saugman noted in 100 cases in which he succeeded in inducing pneumothorax the depth to which it was necessary to penetrate the chest wall as far as the pleura; and in none of them was it deeper than 3 cm.; in the vast majority it was only between 1.5 and 2.5 cm.; in some less than 1.5 and in one even less than 1 cm.

Technic of Insufflation.—As soon as the lumen of the needle penetrates the costal pleura, and there are no adhesions at the point of penetration, the fluid in the closed limb is seen to be sucked up; it rises more or less high. In some cases the suction is so pronounced that the fluid shoots up to the upper end of the tube and care must be taken that it is not aspirated into the pleura. Usually it is elevated between 1 and 6 cm. and oscillates. The patient is told to take a deep breath, and it will be observed that during inspiration the negative pressure is more pronounced than during expiration. This oscillation is the only reliable indication that the lumen of the needle is in the pleural cavity, but at times there are observed slight oscillations when the needle reaches the costal pleura before puncturing it, owing to the repiratory movements of the lung. But these oscillations rarely exceed 1 cm. and must not mislead us. *Only when the negative pressure exceeds 3 cm. may we venture to let in the gas, and beginners should not do it with less than 5 or 6 cm. negative pressure.*

Manometric Hints.—The manometer is to be watched, especially during the first operation. The following are useful guides.

When the Lumen of the Needle is in the Thoracic Wall.—So long as it is outside the endothoracic fascia, the manometer rests at zero.

When it reaches the endothoracic fascia, feeble oscillations, due to respiratory movements of the pleura, may be seen, but they are of slight amplitude, between 0 and 3 on each side of the manometer. They should not mislead us into the belief that the lumen is in the pleural cavity. The fact that there is no negative pressure proves this. In very rare instances, while the needle is yet in the chest wall, a sudden or slowly rising positive pressure is noted in the manometer. It is an indication that the lumen of the needle has entered a large bloodvessel, usually the intercostal artery. The needle should then be quickly withdrawn and the punctured point compressed with the finger. No harm results from it.

A slight negative pressure during inspiration, becoming less on expiration, may be produced when the point of the needle is really not in the pleural cavity at all, but pushing the parietal pleura before it. The indications are clear—the needle is to be pushed ahead guardedly until it punctures the parietal pleura. Allowing gas to enter at this point may produce subfascial emphysema (see p. 820).

After the Needle Has Passed the Parietal Pleura.—When there are no adhesions there is at once seen negative pressure, 5 to 10 cm., and distinct respiratory oscillations, higher on the side of the manometer which is connected with the needle than on the side communicating with the outer air. If the patient holds his breath during inspiration or expiration, or the injection is stopped, the pressure remains negative or positive, respectively.

But at times we meet with this anomalous condition: On passing the parietal pleura the fluid in the manometer rises high, showing negative pressure of 10 cm. or more, but then it remains stationary. We know then that the lumen is in the pleural cavity, and that there are no adhesions, but we hesitate to proceed with the injection because there are no oscillations. It is clear that the lumen of the needle was for a moment between the pleural surfaces, but it has either pushed the visceral pleura ahead of it, or entered the lung, or it has become clogged. In the former case slight withdrawal of the needle will reëstablish oscillations; in the latter case we put the obturator into the lumen of the needle and clear it.

In case there are dense adhesions and the needle does not enter the pleural cavity, the manometer stays at zero and does not oscillate; or when slight oscillations are noted they are but 1 or 2 cm. and equal on both sides, or slightly positive.

When there are slight and yielding adhesions, there is feeble negative pressure, about 2 or 3 cm., and slight oscillations. Occasionally the adhesions yield and the negative pressure, as well as the oscillations, suddenly increase. But usually the pressure becomes positive soon after the introduction of some gas, indicating that a gas pocket has been created. During reinflations, sudden drops in the pressure, due to breaking up of adhesions, are more common than during primary inflations.

When the Lumen of the Needle is in the Lung.—The manometric indications will differ according to the structures the needle has penetrated. If it is in consolidated lung tissue there will be no change in the level of the fluid in the manometer; it rests at zero. If the lumen is in a bronchus or bronchiole, there is usually no negative pressure, but there may be slight oscillations of equal excursions. The amplitude of the oscillations will depend upon the character of the respiration, whether tranquil or labored. When the patient speaks, the respiratory effort with a closed glottis produces, while it continues, a greatly increased pressure, greater still on coughing. When the patient holds his breath, in inspiration or expiration, the manometric readings are again zero. In many cases allowing gas to enter the lung is harmless; it passes out through a bronchus. But in rare instances, when the lumen of the needle is in the parenchyma, gas may enter the interstitial tissues and produce interstitial emphysema (see p. 820).

If after inserting the needle during the first attempt at inflation positive pressure is noted during expiration, it is proof that the lumen is in the lung or in a bloodvessel. Occasionally it is found that the gas flows in freely, but the pressure in the manometer does not ascend. This is an indication that gas is escaping as it enters, which could only occur when the needle is in a bronchus and never when it is in the pleura. "If the key connecting with the nitrogen is quickly opened and immediately closed, allowing only a very minute quantity of nitrogen to flow in, the manometer then becomes positive, it is because the needle is in the lung." (Balboni.)

If the lumen of the needle is in a bloodvessel there are no oscillations, but slight positive pressure may be observed; if some blood enters the needle, which is the rule, the pressure will be rising. When withdrawing the needle it will be found that it contains blood, and the patient may have hemoptysis.

Injection of the Gas.—With the assurance that the needle is in the pleural cavity, the tube leading to the gas reservoir is opened and nitrogen allowed to flow in by aspiration, or pressure when necessary. After 100 cc of gas have entered, the manometer is again consulted, and if still showing negative pressure, another 100 cc are allowed to flow in. It has been my habit never to exceed 300 cc during the first operation, although many do not hesitate to introduce two and even three times as much, and some even attempt to secure complete collapse of the lung during the first operation. Murphy advised the introduction of 200 cubic inches (3000 cc) at the first operation, while Forlanini after mature experience advised only 200 to 300 cc. Clinical experience seems to favor smaller quantities as safer, and many unpleasant, often dangerous, symptoms are thus avoided. To change quickly the relations of the thoracic viscera is dangerous. Moreover, when adhesions are present, they may be forcibly torn apart and cause trouble. When extensive and dense adhesions are present, it is often impossible to introduce more than

100 to 200 cc of gas, and the chances of finally securing a complete collapse of the lung are rather slim.

On the completion of the operation the needle is quickly withdrawn and the index finger of the left hand placed over the point of the puncture and some pressure applied with a view of preventing subcutaneous emphysema. Finally the small wound is sealed with some cotton and collodion and the patient is warned against coughing, which he is to avoid as far as is within his control. I find a dose of morphine or codein is useful for this purpose. It has been my rule to send the patient to bed for twenty-four hours after the first operation, irrespective of his general condition.

Method in Urgent Cases.—In urgent cases, as in copious and uncontrollable pulmonary hemorrhages, and when no apparatus and tank of nitrogen are at hand, we may resort to Murphy's method, which he describes as exceedingly simple: "Take an ordinary hypodermic needle, rub the sharp point dull on a brick, cover the butt end of the needle with cotton, which will serve as a filter of the air that is to enter, then insert the needle into the pleura at the point of election for the production of a pneumothorax. The skin should have been painted with iodine and punctured with a tenotome. The idea is to let the air enter the pleural cavity through a needle, the cotton filtering it as it enters, thus producing a pneumothorax. The finger placed over the butt end of the needle serves as a valve. As the patient inspires the finger is lifted off the needle to allow the air to enter, and on expiration the opening is closed with the finger. In that manner you can pump the pleural cavity full of air to any desired degree of compression. If the patient becomes too cyanotic, or if the breathing is embarrassed, lift the finger from the needle and allow a little air to escape. The procedure is now reversed. Close the end with the finger on inspiration and remove the finger on expiration, so that air will be pumped out instead of in."

Technic of Refilling.—The introduction of a few hundred cubic centimeters of nitrogen does not collapse or immobilize the lung. This must be accomplished gradually by further inflations. In cases with free pleuræ this is a simple matter considering that a pocket with gas has been already created and the needle can be easily introduced into it. For this reason it is best to do the second inflation in the neighborhood where the first puncture was successfully made, so that it enters the gas pocket, and only exceptionally is another place chosen. In the latter case we are guided by the same principles as during the primary puncture.

One thing is to be remembered: The manometer is always to be consulted before the gas reservoir is opened and, in case no respiratory oscillations are seen, the stylet is to be inserted into the needle on the assumption that the lumen may be clogged, which is often the case. If no oscillations are even then observed, the needle is to be withdrawn and reinserted in another place. Accidents have happened during later inflations just as during primary operations.

The quantity of nitrogen introduced during refills depends on the case. My way has been to introduce between 300 and 600 cc at the second and 800 to 1200 at the third operation, provided the patient bears it well. But when I find embarrassment of the circulation, dyspnea, or pain in the chest, I proceed slower and am satisfied with 300 cc given every other day until complete collapse is attained in two or three weeks. We are also to be guided by the final pressure after each inflation. In many cases we get positive pressure after several hundred cubic centimeters of nitrogen have been introduced, although there is no complete collapse of the lung. We often meet with cases in which the gas opens but a small pocket in the pleura and when this is filled the negative pressure decreases or vanishes. When oscillations are good the pressure may be increased guardedly, consulting the manometer after each 50 or 100 cc have entered. Saugman, whose experience is unexcelled, found that if the gas does not pass with 10 to 15 cm. water pressure the case may be given up, because higher pressure will meet with failure.

At times it is noted that during a refill the pressure suddenly sinks. This is an indication that some adhesions have yielded or, which is fortunately exceedingly rare, that the lung has ruptured and the gas escapes from the pleura into a bronchus. This may occur when the nitrogen is introduced under high pressure and the patient coughs vigorously.

My experience coincides with that of Saugman to the effect that it is best that, during the first few fillings, the final pressure should not exceed 0.5 to 2 or 4 cm. of positive manometric pressure. The condition of the patient, as well as his reaction during the succeeding few days should, however, be our guide. We must always watch whether our aim is not attained with a low pressure, and in many cases 0.5 to 1 cm. above zero is sufficient. Forcible inflations involve rapid dislocation of the mediastinum and injury to the other lung. We must bear in mind that it is not always imperative to compress the lung. In most cases affording rest to that organ by immobilization is sufficient to give relief, and this can be attained without high intrapleural pressure. But in case the patient is not improving, his cough, temperature, expectoration, etc., are not influenced favorably, the pressure is carefully and guardedly increased. A final pressure of 10 to 15 cm. of water is too high, though many authors state that they have resorted to it in some cases. Of course, as a rule, the gas is quickly absorbed and within a few days the pressure drops so that the embarrassment of the respiration and circulation is ameliorated. The great problem is presented in cases in which an incomplete pneumothorax has been created and the stiff, unyielding walls of cavities, or dense pleural adhesions, prevent the compression of the part of the lung which we aim to collapse. Saugman and Forlanini have not hesitated to increase the pressure in these cases to 30 and even 40 cm., and they were occasionally rewarded by finally attaining a complete pneumothorax.

Frequency of Refilling.—After complete collapse of the lung has taken place the frequency of the refillings is diminished. In some patients the gas is absorbed slower than in others and we are unable to say in advance who is likely to need frequent refills and who is likely to need infrequent refills. It seems that those walking around absorb the gas sooner than those who remain in bed. A healthy pleura absorbs more air than one that is inflamed. Primarily the guides for the necessity for refills are the general condition of the patient and secondarily the findings on physical examination. An elevation of temperature, if not due to an impending or actual pleural effusion, is often removed by a refill. The same is true of cough and expectoration. In those who have the lung completely collapsed, there is complete absence of breath sounds and adventitious sounds; a return of these is an indication that refilling is necessary. The fluoroscope is, however, the best guide. But I want to repeat that dyspnea and tachycardia, which are often caused by excessive pressure in the pleural cavity, are to be guarded against. Likewise, excessive pressure in the left pleural cavity depresses the diaphragm and may result in gastric disturbances, such as anorexia, vomiting, etc., and concomitant poor nutrition.

The volume of the air within the pleura is influenced by the external atmospheric pressure, which depends on altitude. When a patient in whom a pneumothorax has been induced while he lived at sea level moves to a region of 5000 to 6000 feet above the sea level, the volume of the air in his chest increases in volume about 15 to 20 per cent, and the reverse. To put the matter more concretely: When a patient in New York City with a pneumothorax containing about 3000 cc of air goes to Denver, Colorado, the volume of the air in the chest will increase 15 per cent, or 450 cc. Conversely, when the pneumothorax was created in Denver and he goes to New York, it will decrease 450 cc in volume. For this reason, when a patient has been given an injection of air which filled his chest while he lived at sea level, he should not be sent at once to a high altitude. Conversely, when the patient comes from a high altitude to a low one he needs a refill as soon as he arrives.

Symptoms.—The acute and urgent symptoms of spontaneous pneumothorax are never seen in the artificially created pneumothorax, excepting, of course, when the lung is penetrated and the spontaneous variety complicates matters. The pain, dyspnea, cyanosis and collapse are never encountered. In fact, the majority of patients who have overcome the fear for the operation are ready and well able to leave their beds immediately after the operation and attend to their affairs. The slight difficulty in breathing, seen in some cases at that time, is usually objective, the patient protesting that he feels well although he evidently suffers from air hunger of some degree. But even this disappears within a couple of days, as has already been mentioned. Only in rare instances, when the gas separates adhesions by

high pressure, does the patient complain of pain in the chest, which is, as a rule, trifling.

In febrile patients the effects of the pneumothorax are usually striking, especially when complete collapse of the lung is attained. The fever disappears and, in successful cases, does not return unless there is some complication. The temperature chart (Fig. 124) distinctly shows the effects of collapse on the fever. In some cases it is noted that the fever increases 1° to 3° F. for twenty-four hours after each insufflation (Fig. 125), just as is the case with the reaction after an injection of tuberculin. This is probably caused by increased toxic absorption, owing to the compression of the diseased lung. In case an increase in the temperature, lasting several days, is noted during the treatment, we may look for some unpleasant complication, especially a pleural effusion. When the pneumothorax does not reduce the temperature, we may consider the treatment a failure in this particular case. With the disappearance of the fever, the nightsweats vanish and this gives the patient great relief.

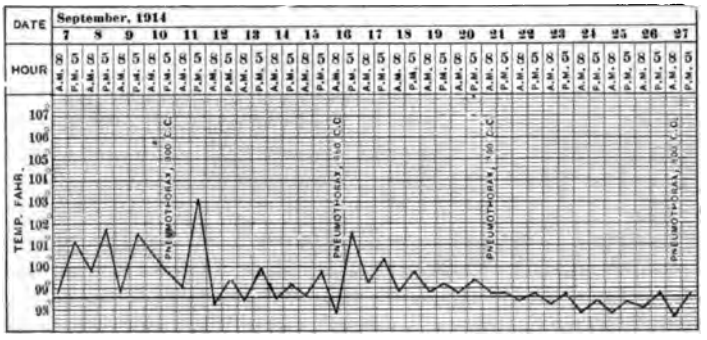

FIG. 124.—Showing the influence of therapeutic pneumothorax on the temperature.

The appetite improves in successful cases, and with this the lost strength is gradually regained, and the languor, which is such a strong clinical feature of the disease, is replaced by a feeling of well-being.

It is noteworthy that, in spite of the improvement in the general condition of the patients, a gain in weight is not a constant phenomenon in artificial pneumothorax. So long as the general condition of the patient is good, and the loss in weight inconsiderable, it should not trouble us. When, however, the loss of weight is considerable and general symptoms, such as fever, sweats, anorexia, etc., make their appearance, we may first try to reduce the pressure in the thorax, and if this does not ameliorate the condition, the treatment may have to be given up.

Great relief is usually obtained in patients who suffer from severe coughing spells which keep them awake during the night. This is especially true of unilateral cases in which a large cavity is emptied

by compression. After the first three or four inflations it is constantly observed that the amount of sputum expectorated is augmented because the pressure exerted by the gas empties cavities and bronchi of their contents. After the lung has completely collapsed, or the cavities have been emptied in partial pneumothorax, the quantity of sputum diminishes, and in unilateral cases expectoration ceases altogether. In many cases tubercle bacilli are not found in the sputum after the lung has been compressed for two months.

The effect of a therapeutic pneumothorax on the blood picture was studied by Gutstein,[1] who found that in cases of complete and uncomplicated pneumothorax the number of red cells increases rapidly, and the percentage of hemoglobin rises slowly. The total number of leukocytes decreases, the lymphocytes increase, and eosinophilia occurs. When the collapse of the lung is not complete, and when there are complications, these blood changes are not constant, and in many cases the reverse was observed, a decline in the percentage of hemo-

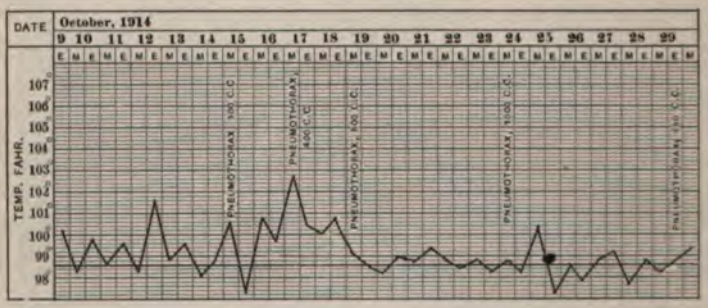

FIG. 125.—Showing the influence of therapeutic pneumothorax on the temperature.

globin, increase in the number of leukocytes, and a decrease in the number of lymphocytes. The blood picture may be used for prognostic purposes: when it shows improvement, the case is progressing favorably, and the reverse.

More striking than the improvement in the general condition is the cessation of hemoptysis when the first inflation is made in a case of hemorrhagic phthisis in which the patient is in constant dread lest the hemoptysis recur. If we succeed in collapsing the entire lung, the hemorrhage ceases at once. The intrapleural pressure acts here like a tampon in uterine hemorrhage. But in some cases the area of lung tissue containing the bleeding vessel is adherent to the chest wall and the intrapleural pressure is powerless to collapse the rigid walls of the bleeding cavity. The result is that the bleeding continues. On the other hand, in some cases we obtain but partial collapse of the lung, but the part containing the bleeding vessel is compressed,

[1] Ztschr. f. Tuberkul., 1916, **26**, 336.

and the hemorrhage is thus stopped. Hemorrhages may occur in patients with pneumothorax. As a rule the blood is derived from the untreated lung, but there are exceptions. The uncollapsed cavity may bleed. At times an increase in the pressure may help in the last class of cases.

In many, though not in all cases, there occurs some dyspnea during the operation or immediately after. But this is, as a rule, transitory. In fact, when the dyspnea is due to fever or toxemia it disappears after the induction of pneumothorax. If excessive pressure is permitted to prevail in the treated pleura, dyspnea is likely to occur which is usually transitory. The absence of the dyspnea, despite the cutting of the breathing area in nearly one-half, is not surprising because, in pneumothorax and in pleural effusion, a reduction of 66 per cent of the respiratory area does not materially alter pulmonary ventilation, nor the chemistry of respiration, provided the patient is at rest.

It appears that a human being can live on much less than two-fifths of the normal breathing area in the lungs. Some years ago S. J. Meltzer[1] called attention to the factors of safety in animal structure and economy, to the extravagance of Nature in furnishing most of the vital organs with a large surplus of tissue above the amount absolutely necessary to perform their physiological functions. Life may continue even when the greater part of the lung is destroyed, provided the disease which caused the destruction is arrested. We see this in cases of pneumonia, pleurisy with effusions, etc. In cases of pneumothorax J. H. Means and G. M. Balboni[2] found that while the patient keeps at rest, respiration, gaseous exchange, carbon dioxide tension, and the mechanical factors are normal. The ventilation of the blood is accomplished almost normally despite the fact that one lung is out of commission. It is for this reason that patients with pneumothorax are dyspneic only on exertion.

Physical Signs.—Recalling the physical diagnosis of pneumothorax as given in text-books, we are surprised that most cases of artificial pneumothorax show but few of the supposedly pathognomonic signs. Thus, tympany is not a constant sign; in some cases the treated side of the thorax is simply hyperresonant and, in contrast with the untreated side, only shows a tympanitic overtone, because of the vicarious emphysema in the latter, which is hyperresonant or even tympanitic on percussion. It is hazardous to diagnose pneumothorax on signs obtained by percussion alone. The only feature that may give a clue is displacement of the heart, especially in cases of left-sided pneumothorax, in which even a small amount of air may shift this organ to the right.

When there is complete collapse of the lung topographical percussion will show all the lung margins far out of the normal. Krönig's resonant field of the apex is abnormally wide; the lower margin is

[1] Harvey Lectures for 1906–1907, p. 170.
[2] Jour. Exper. Med., 1916, **24**, 671.

low, so that the hepatic dulness in the right side, or the splenic dulness in the left, is obliterated. But, what is of more significance, the tympanitic sound may be audible far out beyond the sternum on the untreated side because of "ballooning" (see p. 815), and the cardiac dulness may also disappear. As was already stated this occurs more often in left-sided pneumothorax. In fact, a small amount of gas may have this effect in left-sided pneumothorax, and may be considered a good sign that the operation was successful. In some cases of complete pneumothorax the cardiac dulness disappears altogether.

On auscultation, when complete collapse has been attained, we find total absence of breath sounds, as well as any adventitious sounds which may have been audible before the gas was introduced. In these cases we may be guided by the auscultatory findings as to the necessity for refillings. When the breath sounds are again audible it means that a considerable portion of the air has been absorbed and must be replaced at once. Especially is this true when rales reappear in places where they were audible before the lung was collapsed. However, we meet with cases of good collapse of the lung, but large bronchi remain open, and, under circumstances which are as yet not clear, we hear distinct and exquisite amphoric breathing; at times even metalic sounds; which shows that the teachings of some text-books to the effect that the amphoric phenomena in pneumothorax are invariably due to bronchopleural fistulæ is erroneous. They are evidently due to sounds originating in the bronchi which reverberate in the air-filled pleural cavity. A metallic tinkle is very frequently heard, especially in cases in which bands of adhesions run from the mediastinum to the chest wall.

When such adhesions are thick there may be no changes in the breath sounds at all; they act as good conductors of sounds originating in the bronchi. I have recently witnessed while examining a patient the following phenomena: Metallic breath sounds and a tinkle were distinct. But suddenly the patient went into collapse with dyspnea, cyanosis, tachycardia, cold extremities, etc. Coincident with the collapse, which was evidently due to the rupture of an adhesion, all the sounds in the chest, audible a few minutes before, disappeared. Adhesions may serve as conductors of adventitious sounds engendered in parts of the lung which are not collapsed, as well as from those originating in the opposite lung, as will be shown later on.

The pneumothorax is effective in displacing the mediastinum toward the untreated side. In some patients there is a "weak mediastinum," and the result is that the displacement is excessive, and distress of variable degree may be manifested, such as pains in the chest, dyspnea, insomnia, and at times pseudo-asthmatic attacks. In these cases it may be necessary to withdraw some of the gas and thus reduce the pleural pressure. High pressure may also result in "ballooning" of the pleura toward the untreated side at certain so-called weak spots

which have been described by various roentgenologists, and by Stivelman[1] in this country. The inflated pleura may be seen bulging under and beyond the sternum especially between the first and the third ribs. There are two weak spots in the mediastinum, one above and anteriorly, right under the manubrium and upper part of the gladiolus, the place where during embryonal life and infancy the thymus gland is located. It extends from about the first to the third ribs. In adults this place is filled with connective tissue, elastic fibers, and remnants of the thymus. The second weak spot is found in the lower part of the posterior mediastinum, in front of the spine and aorta and behind the esophagus and heart. Under normal conditions the right pleura extends to the middle line, because the aorta and the esophagus are located somewhat to the left and prevent the encroachment of the left pleura to the right side. With excessive pressure in a pneumothorax, and in some patients with a weak mediastinum even with moderate pressure, there occurs ballooning of the pleura at either of these two points, at times to an extent as to make it appear almost as if there were mediastinal or pleural herniæ. Anteriorly the pleura may encroach upon the opposite side, under the sternum, and even beyond it. Posteriorly ballooning is quite common in right-sided pneumothorax, while in left-sided pneumothorax it is rare for the above-mentioned anatomical reasons. Roentgenograms may show this hernia as a bright area with a sharp outline. At times it may be discovered by percussion by its hyperresonant or tympanitic note at the side of the sternum; in rare instances rales which are engendered in it are audible near the sternum or the spinal column of the untreated side, and they may lead to diagnostic error. At times percussion with a coin will bring out an exquisite sound. On the whole, it may be said that slight cases are harmless; but when the hernia is extensive it may lead to irritative cough, distress under the sternum, and compression of the unaffected upper lobe of the lung. Reduction of the pressure in the pleura may have to be resorted to for relief. Roentgenograms of these herniæ are shown in Figs. 3 and 4, Plate XX.

The progress of the pneumothorax can usually be followed by noting the increase in the area of the thoracic surface over which there is either absence of respiratory sounds or amphoric breathing after each filling, until finally the complete lung is collapsed and all breath and adventitious sounds disappear.

Complications.—Not all cases of induced pneumothorax run a smooth course during the period of treatment. Complications may arise during the operation or immediately after, and while the patient goes around with a collapsed lung. Of the former, collapse, gas embolism, pleural shock, or pleural eclampsia, pain in the chest and subcutaneous emphysema are worthy of consideration; of the latter pleurisy, pleural effusion and rupture of the lung are the most important.

[1] Jour. Am. Med. Assn., 1919, **72**, 1445.

Accidents during the Operation.—At times, when the needle is inserted rather high in the intercostal space, a bloodvessel is injured and bleeding results. In most cases it is insignificant, but I have seen a hematoma about one inch in diameter result. When there is fluid in the pleural cavity such bleeding may turn it sanguineous. We have not observed any serious effects from these accidents.

When the intercostal spaces are narrow, and at times because of carelessness, a rib is injured by the needle, the patient complains of severe pain, but we have not observed the development of periostitis as a result of this accident. Even in cases in which there is pus in the pleural cavity, pyopneumothorax, injury to a rib with the needle does not produce periostitis, though fistulous tracts result very frequently.

Penetration of the visceral pleura and perforation of the lung occurs very frequently, as is stated elsewhere. It is harmless, as long as the puncture occurs into healthy lung tissue. The most that can happen is sanguineous expectoration for a few hours. This is true when the needle is directed vertically downward and not obliquely. In the latter case the lung may be lacerated, not only punctured, and the leakage of air into the pleura is so pronounced as to produce complete collapse of the lung without any air entering from the gas bottle. Even this is harmless, as a rule, as long as the laceration has occurred in healthy lung tissue. When the puncture is in diseased lung tissue, the result is pyopneumothorax, a grave complication, from which very few patients recover.

Pleural Shock.—Pleural shock may be of various degrees. The mild forms manifest merely an increase in the rate of the pulse and respiration, pallor, dyspnea, etc., which pass within a few minutes or an hour. I have met with it several times; in one patient it occurred consecutively during the first four inflations and I am inclined to attribute it in a great measure to his fear for the operation. In one of my cases the shock was quite severe, even alarming, yet it passed away within half an hour. Several authors have reported fatal cases.

The etiology, especially of the fatal cases, is not clear. Forlanini, Saugman and others are inclined to attribute it to reflex spasm of the cerebral or cardiac bloodvessels. It has been observed that thoracocentesis for any purpose may cause collapse or even death in very rare instances. Brauer is inclined to attribute the symptoms of shock to gas embolism in most cases and says that the fact that it is usually transitory does not exclude gas embolism. But pleural shock may occur without any gas inflations. James A. Lyon[1] mentions a case occurring while injecting novocain into the pleura.

That this accident is comparatively rare is evident from Forlanini's figures to the effect that operating on 134 patients, not including those in whom he failed to produce a pneumothorax, and making more than 10,000 operations, he met with pleural shock only twelve times.

[1] Boston Med. and Surg. Jour., 1914, **171**, 329.

Among more than 3000 inflations made at the Montefiore Hospital we observed it but twice to be sufficiently severe to cause some alarm.

Gas Embolism.—When the manometer is not properly consulted, it is said that at times even when the most careful technic is followed, gas may enter a bloodvessel and be carried to any part of the body and produce an embolism. Usually one of the pulmonary veins is entered; it is well known that negative pressure prevails in these vessels. Brauer maintains that one of the veins around an infiltrated area of lung tissue, or of pleural adhesions, may be penetrated by the needle and gas introduced into the circulation. The nitrogen is carried into the left heart, then into the aorta, whence it may travel into the coronary arteries or the cerebral vessels. Experimental researches have not been uniformly confirmatory of this theory, and clinically the symptoms of embolism have been observed in some cases even when no nitrogen was allowed to enter through the needle—merely after introducing the needle.

Wolff-Eisner,[1] while agreeing that in most instances it is due to gas embolism, says that there are some in which thrombi are responsible for the symptoms observed. They are derived from the vessels around or within the pulmonary or pleural lesion, and dislodged by the needle. However, it must be emphasized that symptoms of gas embolism are not exclusively encountered in the primary operations, but have been met with during refills.

In many cases gas embolism is difficult of diagnosis. The symptoms of pleural shock simulate it to a degree as to render the diagnosis doubtful in many instances. It is, however, to be remembered that pleural shock occurs during several operations in a given patient; in some, until they become convinced of the harmlessness of the procedure, while gas embolism occurs but once, and is rarely repeated. It has been stated that in gas embolism there may be found gas bubbles in the retinal vessels. But this must be very rare, because in some fatal cases of gas embolism the autopsy failed to disclose the gas within the bloodvessels.

The symptoms are collapse, rapid pulse, irregularity of respiration, numbness, giddiness, inequality of the pupils, hemiplegia, etc. In some rare cases death has occurred without warning. I have been fortunate in not having met with a single case of this kind in my practice. Of course, prophylaxis is to be the chief aim while operating, and one who does not permit the gas to flow into the chest without considerable oscillations of the manometric column is hardly likely to meet with a case. Fatal cases have, however, been met by the best and most experienced operators.

The treatment of gas embolism must be prompt and vigorous. Cardiac stimulants, camphor in oil hypodermically, digitalis intravenously, oxygen inhalations, and artificial respiration must be given at

[1] Die Prognosenstellung bei der Lungentuberkulose, Berlin, p. 498.

once. Jessen advises venesection, and Neumann uses intravenous injections of adrenalin.

Pains.—Pains in the chest are felt by the patient occasionally during the operation. At times, while introducing the needle as far as the costal pleura, and before penetrating it, exquisite pains are felt which promptly disappear as soon as the pleura is punctured. This can be prevented by proper anesthesia of the pleura with novocain or cocain. Very often after the introduction of the gas, pains are felt in the chest for twenty-four hours, due to breaking up of adhesions, especially when high pressure is applied. They are not at all unbearable and need no treatment. Abdominal pains may result from lowering of the diaphragm by the intrapleural gas pressure, but this is also transitory and needs no treatment.

Spontaneous or Pneumogenous Pneumothorax.—Spontaneous pneumothorax may occur when the needle lacerates the visceral pleura, or when a superficial lesion or cavity of the lung breaks through after the pleural sheets are separated by the gas. Forlanini met with 9 cases of this kind. Floyd[1] and Webb[2] mention it. Meyer[3] mentions a case in which it occurred while preparations were being made for the induction of an artificial pneumothorax. According to Brauer and Spengler[4] a spontaneous, or pneumogenous pneumothorax may occur when the needle happens to strike an emphysematous bleb, and they mention a case in which an autopsy confirmed this opinion. Wallgren[5] has met with 2 similar cases. Of course, when this complication is due to the entry of the needle into a cavity, or a caseating part of the lung, perforation of the lung, with its concomitants, is likely to be the result.

According to W. Parry Morgan, "spontaneous" pneumothorax is more often produced while inducing an artificial pneumothorax than is generally appreciated. This is confirmed by the occasional cases met with in which the treatment is abandoned after a futile attempt to introduce gas into the pleura, and a collapsed lung is then discovered. Again, a roentgenogram of the chest taken after the first operation usually shows evidence of more gas in the pleural cavity than has been introduced from the reservoir. While it is common experience of those using the method that gas can be detected after 200 to 300 cc have been introduced, it has been Morgan's experience that if the visceral pleura is not injured the gas cannot be detected until considerably more than 300 or 400 cc have been introduced. He concludes that when a pneumothorax is visible in the fluoroscope after introducing 300 or 400 cc of nitrogen, we have justification for the conclusion that roentgenographic demonstration of a pneumothorax after the intro-

[1] Boston Med. and Surg. Jour., 1913, **169**, 713.
[2] Tr. Nat. Assn. Study and Prevent. of Tuberc., 1914, **10**, 101.
[3] Ibid., p. 112.
[4] Handb. d. Tuberkulose, 1919, **3**, 180.
[5] Beitr. z. Klinik d. Tuberkulose, 1916, **35**, 319.

duction of such a quantity of gas is achieved only by this being largely supplemented by leakage from the lung.

On the whole, this complication is harmless in most cases. The patient becomes dyspneic, cyanosed, and looks pretty bad for several hours, a day or two. But then the circulation adapts itself to the altered position of the thoracic viscera, and the patient feels quite well. We then continue the treatment by refills according to indications. In rare instances, when the needle enters a tuberculous cavity or a caseating part of the lung, pyopneumothorax is the result.

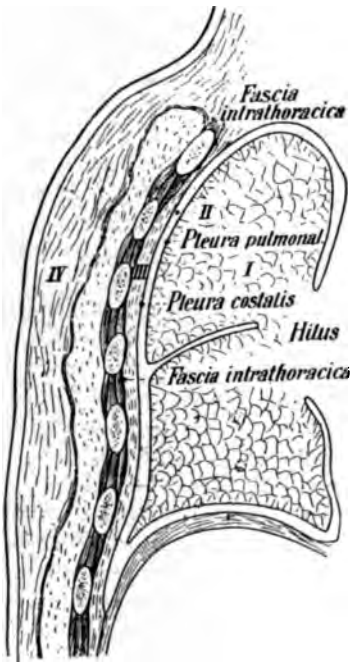

FIG. 126.—The different forms of interstitial emphysema. (After Brauer.) I. Interstitial emphysema of the pulmonary septa; leads to real mediastinal emphysema. II. Adhesion emphysema. III. Subfascial emphysema, which may extend to the neck and to the opposite side. IV. The most common form, emphysema of the subcutaneous and submuscular tissues.

Emphysema.—The infiltration of gas into the subcutaneous tissue of the thoracic wall around the point of puncture is very frequently observed, especially in those operated upon by the Brauer method. In the vast majority of cases it is due to the high pressure of the gas in the pleural cavity, supplemented by cough, and the air works its way along the track of the puncture. It is readily recognized by the crepitation elicited on palpation, and is of little significance—passing away spontaneously within three or four days or at most a week, and further inflations are not contraindicated while it is present. It may

be prevented by using thin needles, and warning the patient against coughing, or administering some sedative like codein immediately after the operation. It has occurred in about one-half of my cases after the first or second operation and rarely after later inflations.

Of more serious import is emphysema of the deeper tissues of the thorax, which, fortunately, occurs only rarely and may be avoided by careful technic. It is usually due to the introduction of air into the subpleural tissue before the lumen of the needle has penetrated the costal pleura. When gas enters between the pleura and the intrathoracic fascia, so-called *subfascial emphysema* arises. The air in this form of extra-pleural emphysema oscillates with the respiratory motions of the chest. When the needle reaches that space and the operator is under the impression that it is in the pleural cavity and permits further entry of air under pressure, the parietal pleura may be separated from the chest wall, and air enters between it and the intrathoracic fascia and proceeds upward to the neck producing subcutaneous, or submuscular emphysema. The separation of the parietal pleura and the appearance of air in the neck is often accompanied by more or less severe pain. It may occur soon after the operation, or several hours later.

Deep emphysema may also be due to leakage from the pleural cavity through the wound made by the needle, the gas being pressed by the intrapleural pressure or the respiratory movements especially during cough, into the extrapleural tissues. Saugman is of the opinion that this may even occur without excessive intrapleural pressure, although the latter enhances the chances of its occurrence. The gas works its way along the path of the vessels to the posterior mediastinum and thence along the vessels and trachea up to the neck, where we may discover it by the crepitations along its anterior aspect. It is noteworthy that it is often felt earlier on the untreated side of the neck, which Saugman believes is due to posture. Rarely, the emphysema extends along the vessels to the face, shoulder, arm, and forearm. I have seen cases in which the abdominal wall and even the scrotum were involved. It may be severe enough to cause dysphagia, and pain wherever it occurs. But the ultimate outcome is always favorable—it disappears within a few days or a week. It has occurred in several of my cases and, barring the little inconvenience it caused them, it was of no significance. But Jessen[1] reported a case in which mediastinal emphysema occurred during the first puncture and the patient died from asphyxia. Saugman, who had considerable experience with deep emphysema, states that in the patients in whom it occurs there are but few chances of inducing a complete pneumothorax because of the gas leakage.

In rare cases the needle hits an adhesion, or penetrates the lung, and air is thus allowed to enter the pulmonary tissue, resulting in *interstitial*

[1] Deutsch. med. Wchnschr., 1913, **39**, 1245

emphysema. Because the air is, in these cases, within the visceral pleura it cannot wander up to the neck, as is the case in subfascial emphysema. In both cases, however, the air may be responsible for a tympanitic note on percussion and mislead into the belief that a pneumothorax has been created.

Pneumoperitoneum, which has been described by several authors, I have observed but once. It may occur when the needle is inserted along the lower margin of the chest and the diaphragm happens to be unduly high, which is not unusual in pulmonary tuberculosis. The lumen of the needle may then reach the peritoneum, between the diaphragm and the stomach or liver, producing subphrenic pneumothorax or pneumoperitoneum. It is to be remembered that there also the manometer will show negative pressure, oscillating with the respiratory movements. It is difficult to distinguish these oscillations from those seen when the needle is in the pleural cavity, but if it is carefully watched it will be observed that when the needle is in the pleural cavity the negative pressure is stronger during inspiration, and the reverse is true when the lumen of the needle is in the peritoneal cavity. In the case that came under my observation, the house physician reported to me that in a patient who had adhesions of the pleura which prevented me from introducing gas, he succeeded in getting into his pleural cavity about 1000 cc of air. But the patient stated that he had pain in the abdomen, and that he felt as if the air had entered his "stomach." An examination showed that the abdomen was blown up, highly tympanitic on percussion, and the roentgenogram showed distinctly gas in the peritoneal cavity. He made an uneventful recovery, the air being absorbed within a few days.

Pleurisy.—Dry pleurisy occurs very frequently in patients with artificial pneumothorax. In many cases it is followed by effusions, but in some it remains dry and the symptoms pass with a few weeks. It may follow exposure to cold, or any of the acute inflammatory conditions of the nose and throat. Irregular fever and pain in the chest are the most common symptoms. Hyperalgesia of the chest wall is another very frequent symptom; pain is elicited on pressure of the intercostal spaces. Patients who ceased coughing since the collapse of the lung begin again to cough spasmodically and painfully, while the dyspnea becomes more pronounced. Physical examination may not elicit any signs, but in those in whom there is no complete collapse of the lung we may hear a friction sound which disappears with a change in the position of the patient, or after the pleura is refilled with gas.

Pleural Effusions.—The most frequent and serious complication of artificial pneumothorax is pleural effusion in the course of the treatment. Its frequency varies with the different reports by various authors. Some report as high as 70 per cent of cases, while others have met with it less frequently. It appears that those who report a low percentage of incidence refer to cases which had not been

followed to termination. Some are inclined to attribute it to "colds," or to "rheumatism," etiological factors which are open to question. Others have stated that it is usually due to infection during the operation, and maintain that when asepsis is rigidly observed effusions are rare, which does not hold, because effusions have been met by the most careful of operators. Many authors consider the nitrogen as a foreign body which irritates the serous surface of the pleura, predisposing it to disease. It becomes a *locus minoris resistentiæ*, and inflammation occurs more easily than in ordinary cases of phthisis. Klemperer's[1] explanation is more plausible: Disease processes which reach the surface of the lung and the visceral pleura cause adhesions in patients with normally superimposed pleural sheets, but in pneumothorax with separated pleural sheets exudative inflammations are the result. Rupture of adhesions which lay bare tubercular foci in the pleura may also be instrumental in infecting the complete serous surface.

There are observed three main groups of effusions complicating therapeutic pneumothorax:

1. Purely serous with a poor cytology, and hardly ever abundant. They are at times not even recognized clinically, and, after remaining for a few weeks, they are absorbed without leaving any evident traces behind.

2. Serous effusions with a large number of cells, which have a tendency to increase in size so as to almost fill the chest. Their symptomatology is clear cut, producing, as they do, high fever lasting for weeks and interfering with the successful outcome of the case. In most of these effusions large numbers of tubercle bacilli may be found microscopically, or by inoculation experiments. At times, they become purulent.

3. Effusions due to mixed infection, of exogenous or endogenous origin. In the former case the infection is brought about by an unsterilized needle, in the latter, by pyogenic bacteria derived from the lung coming into the pleura through the injuries produced separating the adhesions. Intercurrent respiratory diseases, tonsillitis, bronchitis, influenza, etc., may also be held responsible in exceptional cases.

Brauer and Spengler[2] maintain that some exudates convey the impression of transudates, especially when they appear while the lung reëxpands and strong intrapleural negative pressure is induced.

Symptoms.—In many patients with pneumothorax there occur small effusions which give no symptoms. Because of the small amount of fluid in the pleura, it is not recognized clinically; neither physical examination, nor roentgenography discloses their presence. In some patients these effusions are discovered accidentally. In others, there is slight fever and discomfort for a few weeks, but then the temperature declines and the patient keeps on doing well. There is, however, one

[1] Berl. klin. Wchnschr., 1911, **147**, 372.
[2] Handb. d. Tuberkulose, **3**, 225.

point which may give a clue. Inflammatory conditions of the pleura reduce its absorptive powers and refills are not needed as often as when the pleura remains normal. If the intrathoracic pressure rises, and overinflation cannot be considered a cause, an exudate is usually the cause. The fluid in these cases is clear, and is poor in cells, only lymphocytes are found in small numbers.

In others the onset is stormy, with pain in the chest, fever and prostration. An examination at this early stage may not disclose any signs of an effusion. But it has been my experience that when high, continuous, or irregular fever suddenly appears in a patient with an artificial pneumothorax who had been doing well before, there is an acute pleurisy, and that signs of an effusion will be discovered within a few days. It will, however, soon be noted that the intrapleural pressure increases. In many cases the fever declines within two or three weeks, dropping down to the level at which it was before the complication ensued. In some, the fever continues for months, though no signs of mixed infection are discovered. In the afebrile stage the exudate may remain within the chest for many months; I have seen a case in which it remained for over three years. But the patient was doing comparatively well, gaining in strength and weight. Microscopical examination of the exudate shows that the number of lymphocytes is large, though in many polynuclear elements are in abundance. If the exudate remains for a long time it may become cloudy and greenish in appearance and polynuclear lymphocytes, as well as eosinophiles are found microscopically. Even then, there may be no other microörganisms than tubercle bacilli in the exudate. In some cases, after remaining purely tuberculous for some time, mixed infection takes place, and then they belong to the group of septic pleurisies. After the acute stage has passed, the patient may feel quite comfortable for a long time, though the fluid in his chest remains quite abundant, excepting for the dyspnea on exertion and precordial heaviness which trouble him.

The third group consists in septic exudates, which are fortunately not common. The onset may be even more stormy than in the preceding group. In some, a chill occurs, followed by high fever; 103° to 104° F. is not exceptional. The pains in the chest may be severe. For a time the fever is continuous, or remittent, but later it becomes hectic, with chills and profuse sweating. It is noteworthy that death during the first few weeks is rare unless the effusion is due to perforation of the lung (see p. 827). As a rule, these patients last for months despite the fever and concomitant symptoms. In some, the fever abates, dropping to 100° or 101° F. and the patients live for months with pus in the pleural cavity. One of my patients lived thus for three years, was quite active at his vocation, when the pus began to point on the chest wall. An operation gave him relief for another year, when the pus again began to point on the chest wall, and he recently was operated upon again, but the operation disclosed that there was merely a cold abscess

of the chest wall. In the majority of cases, however, the fever persists, tuberculous changes in other organs, notably the larynx, intestines, etc., make their appearance, and finally amyloidosis of the visceral organs helps in ushering out the patient. It is noteworthy that removal of the pus during the acute stage may increase the fever by favoring absorption of the toxic products; this may happen even after an exploratory puncture.

The bacteriology of these purulent effusions is that of empyema in general. In milder cases pneumococci and other diplococci are found; in the severer and toxic types, staphylococci and streptococci and, at times, hemolytic forms of these microörganisms. As is common in mixed infections, the tubercle bacilli are outnumbered by the pyogenic germs. The cytology is also that of a severe infection—a high count of polynuclear lymphocytes, etc. The blood changes are those of pyogenic infections.

Pleurisy with effusion in the untreated side is extremely rare. So far only 6 cases have been reported by Als,[1] Fishberg,[2] Stivelman,[3] Reynier and Rossel,[4] and Zemmin[5] of pleurisy with effusion in the untreated side with a hydropneumothorax after artificial collapse of the lung. In the case reported by the writer, a hydropneumothorax was complicated by an effusion into the left pleura. Both effusions were absorbed within three months, and the patient made an uneventful recovery.

Small effusions are often very difficult to diagnosticate, and even roentgenography may fail to reveal them; they are especially liable to be missed on a plate which was taken with the patient in a reclining position, for obvious reasons. In the erect position careful examination of the costopleural sinuses will usually reveal them, especially in the left pleura, when the patient is bent toward the affected side while he is viewed in the fluoroscope. When more or less copious, the usual signs of hydropneumothorax, flatness, absence of breath sounds, succussion, and a splash are elicited. The flatness elicited on percussion shifts with the position of the patient. Now and then, exquisite metallic sounds, and a metallic tinkle are heard on auscultation.

Effects of the Pleural Effusion.—The effects of these effusions depend on two main factors: Whether they are copious, and whether they are septic. Some authors have stated that small serous effusions are rather salutary phenomena, and that they may have a good effect on the tuberculous process by the antibodies they produce (see p. 515). But personal experience has led me to the conviction that all effusions, small as well as copious, turn out unfavorable in most cases. Even small effusions reduce the absorptive powers of the pleura, and have a

[1] Ztschr. f. Tuberkulose, 1920, **31**, 333.
[2] Am. Rev. of Tuberc., 1920, **4**, 659.
[3] Am. Jour. Med. Sci., 1921, **162**, 270.
[4] Rev. Méd. de la Suisse Romande, 1921, **41**, 169.
[5] Beitr. z. Klinik d. Tuberk., 1921, **47**, 169.

strong tendency to produce adhesions and more or less extensive obliteration of the pleural cavity wherever the two inflamed sheets meet. The clots which form in these exudates, even in small ones, organize and interfere with further inflations of air, and in extreme cases, which are not uncommon, the entire pleural cavity is thus obliterated. The thickened pleura is often felt by the operator who attempts to reinflate the chest as a very perceptible obstruction to the entry of the needle. Later, when these adhesions are organized they embrace other organs, especially the trachea, mediastinum, and diaphragm pulling them toward the affected side. Lasting for a long time they produce secondary shrinkage of the bony thorax, manifesting itself as flattening and retraction of the affected side, the ribs approach one another, in extreme cases even overlapping one another, the shoulder droops, and the spine bends; the scoliosis having a concavity toward the side with the pneumothorax.

Some writers, in this country B. Stivelman,[1] say that repeated aspirations of the fluid, especially after watching its effects on the position of the mediastinum, and refilling the cavity with air, will prevent adhesions. In my experience, when adhesions begin to form, we are unable to prevent them from nullifying the good effects of the pneumothorax by any known means. Sooner or later it becomes more and more difficult to refill the pleural cavity with gas, and the treatment must be given up.

Treatment.—So long as there is no high fever, and especially no cardiac embarrassment, it is best to leave these effusions alone because they keep the lung collapsed, and this is what we aim at with the treatment. Acute inflammatory processes of the pleura reduce the power of absorption of this membrane, and refills are not needed as often as when it remains normal.

But in some cases in which the fluid in the pleura accumulates rapidly and induces dyspnea, cyanosis, etc., or when the fever and prostration are marked, it may be necessary to withdraw part of the fluid and replace it with air, leaving the final pressure at 0, or even below. In the usual case of this kind it is preferable to withdraw the fluid because we may thus reduce the fever, though many writers state that they prefer to withdraw the gas in cases of cardiac embarrassment. However, it appears that soon after withdrawal the fluid usually reaccumulates. In some cases I have applied autoserotherapy—withdrawing 10 cc of fluid and reinjecting it subcutaneously, and am under the impression that it enhances absorption. Brauer objects to autoserotherapy in these cases on account of the large number of virulent tubercle bacilli in the exudate. But I have never observed a tuberculous abscess at the site of the injection of serous fluid.

These exudates must always be carefully watched. In case they are absorbed too rapidly, the lung reëxpands and may form adhesions,

[1] Jour. Am. Med. Assn., 1921, **77**, 12.

thus preventing its further collapse by gas inflation. I have given a fair trial to the various methods of gas replacement which many authors have suggested, and found them of questionable value. Withdrawing the fluid and injecting gas instead, either in one operation or separately, has not given me the results claimed by some writers. Inasmuch as the fluid soon reaccumulates, the intrapleural pressure increases enormously, and the patient again suffers from cardiac embarrassment. In some cases it is necessary to withdraw fluid or gas soon after a replacement operation. The suggestion that watching the position of the mediastinum will give a clue as to the indications does not hold in most cases. In some it is held by adhesions and even high pressure will not move it much; in others, there is a "weak mediastinum," already mentioned, and it moves with the least amount of gas or fluid in the pleura.

It may be stated again that a copious effusion is a serious complication of artificial pneumothorax. In a large proportion the treatment must be abandoned sooner or later because of adhesions; in many complete obliteration of the pleural cavity results. But even then the patient may do fairly well because the fibrosis with which this process is accompanied extends into the lung and the patient may recover to a degree as to be able to pursue some vocation in which muscular exertion is not essential. In other words, the case becomes one of fibroid phthisis and the prognosis is fairly good.

With septic effusions things are different. Pyothorax complicating artificial pneumothorax is a serious complication. Some last for a long time, but in most cases the fever, emaciation, etc., are instrumental in dragging the patient down hill and, within a few months, he succumbs to exhaustion, amyloid changes in the viscera, extrathoracic tuberculous lesions—of the larynx, intestines, etc. The usual surgical treatment of pyothorax is, in most cases, powerless to check the progress of the disease and most authors prefer repeated aspirations of the pus from the chest, according to indications. Now and then we meet with a case in which the pus remains in the chest for months, or even years, and the patient keeps afebrile. In the septic cases aspiration has its drawbacks: Fistulous tracks are often left at the points where the needle is inserted to withdraw the pus. Several surgeons, notably Sauerbruch,[1] Spengler, and others, report good results with thoracoplastic operations. But even when one of these surgical operations is successful, the patient remains an invalid. Recently I have seen good results with the Carrel-Dakin method of irrigating the pleura.

In rare instances the pus finds its way out through a bronchus and the patient drains it out, spitting large amounts of purulent material for months, and finally recovers. In these cases we should instruct the patient as to the value of posture in facilitating the expectoration of the pus from the chest.

[1] Die Chirurgie der Brustorgane, Berlin, 1920.

Perforation of the Lung.—We have pointed out elsewhere in this book that small cavities in the lung are often located subpleurally, and that caseation and softening of the pleura are not exceedingly rare in pulmonary tuberculosis. So long as the pleural sheets are in apposition, organized adhesions prevent, in most cases, the breaking through of these lesions into the pleural cavity. But a pneumothorax, especially if the gas in the pleura is not at a high pressure, will favor perforation of these lesions with resulting infection of the pleural cavity.

In others, as I have seen at necropsies, the rupture is due to adhesions tugging upon the pleura, especially during cough, overexertion, etc. The tear occurs in these cases at the site of the attachment of the tense adhesion. In some cases it may be said to be due to direct perforation of the visceral pleura with the needle while attempting to fill the pleura. This may be prevented by invariably directing the needle vertically downward, and not obliquely, so that if the visceral pleura is punctured it is not torn. In rare instances perforation occurs through a pleura already filled with pus, and may be considered empyema necessitatis, the pus breaking through the visceral pleura.

Perforation of the lung results in an open pyopneumothorax. But in the spontaneous open pneumothorax the air in the pleura, as well as the elasticity of the lung, are instrumental in closing the opening into the parenchyma, as a rule. Here, however, the perforation remains open indefinitely because of the rigidity of the pleura, as well as of the tuberculous changes in the perforated lung. The result is that all efforts at clearing the pleural cavity of septic material prove unavailing; it is reinfected constantly from the lung lesion. Attempts at refilling are futile, because the gas escapes through the opening into the lung, and the intrapleural pressure is always that of the external air.

Perforation is not extremely rare, and has occurred in some cases in which before the accident the pneumothorax brought excellent results. Burnand[1] reports that among 300 cases of artificial pneumothorax perforation occurred fourteen times, which was about the ratio observed by most authors.

Perforation of the lung occurs suddenly. The patient, who may have been doing well, is suddenly seized with intense pain in the chest; the temperature rises and signs of a pleural effusion soon make their appearance. I have had a case in which the perforation occurred during a refill and I noted that the intrapleural pressure, as registered by the manometer, dropped suddenly. The subsequent course is that of an acutely progressive pyopneumothorax. In nearly all cases the rent in the visceral pleura remains open indefinitely, and the pleural cavity is constantly reinfected from the tuberculous lesion in the lung. In many instances the water-whistle sound may be heard owing to the air rushing in during each inspiration through a bronchus which reaches into the fluid within the pleura.

[1] Ann. de Méd., 1921, **9**, 22.

Those who do not succumb soon to this accident, remain with a chronic pyopneumothorax, but the pus in the chest is drained through a fistula communication with a bronchus. Posture influences drainage. Now and then drainage is interfered with and the fever rises. Sooner or later hectic fever makes its appearance and amyloid changes in visceral organs, the liver, kidneys, intestines, etc., give symptoms. Death is finally due to exhaustion.

The treatment is purely symptomatic. Even in the cases in which the purulent secretions are well drained through the bronchi, recovery is unlikely. The various operative procedures which have been suggested have proved of no real value, though Spengler reports some success attained by repeated aspirations of the fluid and a series of plastic operations. Prophylaxis is, however, the only rational thing to observe. Proper technic in inducing pneumothorax, especially in handling the needle, is of prime importance. Those in whom there is but partial collapse of the lung should avoid overexertion, because of the danger of tugging of adhesions and tearing the lung. Maintaining moderate or high intrapleural pressure is another excellent prophylactic measure. This can be attained by timely refills.

Active Lesions in the Untreated Side.—Extension of the disease in the other lung is perhaps the most disheartening complication during the treatment. It has been stated that it may be caused by an attempt to collapse the more affected lung too quickly; the purulent matter is squeezed out rapidly, and it travels along the bronchi to the other side of the chest, producing pus embolisms. It has also been attributed to excessive pressure in the pneumothorax. It has occurred in some of my cases and in none could I attribute it to these causes. In some of my cases there was a hemorrhage from the untreated lung, but it soon ceased. The writer has had cases in which one side of the chest was treated by a pneumothorax and the lesion was cured, but subsequently a new lesion flared up in the opposite lung, which was also treated by a pneumothorax. This indicates that the collapse and compression of a lung do not necessarily impair its function permanently.

Indications.—Forlanini at first urged that only far advanced cases of phthisis for which everything had already been tried, but no relief was obtained, should be given artificial pneumothorax. As a *conditio sine qua non* it was insisted upon that the lesion must be strictly unilateral, and that any involvement of the other side of the chest is a contraindication to the treatment.

Factors Entering into the Selection of Cases.—*The Form and Stage of the Disease.*—There are numerous cases of phthisis which are doing well and recover, with or without any treatment, medicinal, specific, climatic, or institutional, and it is, of course, not advisable to subject them to the operation with its potential complications. This is true of mild incipient cases, and abortive tuberculosis. Fibroid phthisis runs an exceedingly chronic course; the pleura is often exten-

sively involved, precluding the introduction of gas into the hemithorax most affected, and cannot be treated by this method. This is also true of the most common forms of fibroid phthisis characterized by diffuse fibrosis all over both lungs, and it would be sheer folly to treat but one side of the chest. On the other hand, in the later stages of diffuse fibrosis, when excavations form in one lung, the question of pneumothorax is to be considered, provided, of course, that the pleura is free from dense and extensive adhesions.

It is the acute and progressive form of phthisis in which artificial pneumothorax finds its best indications and shows the most striking therapeutic results. In the group of cases known as galloping consumption, in which the patient is carried off within three to six months by a rapidly progressing infiltration, caseation and excavation, there are many who can be saved by the induction of pneumothorax. It is fortunate that dense pleural adhesions are exceptional in these cases, and a pneumothorax can easily be induced. The results are often astonishing—with the collapse of the lung, the tachycardia, fever, nightsweats, cough, expectoration, etc., disappear, and within a few weeks the patient is reinvigorated and may continue to gain in weight and strength indefinitely.

It must be stated, however, that the ultimate results are not as good in acute as in the chronic cases. Soon after the lung is collapsed the results are marvelous—high fever declines, the appetite returns and gains in weight are registered. But these results are not lasting. Within a few months, owing to some complication, a change takes place much quicker than the initial improvement and the progress is now downward. The best results are, after all, obtained in chronic cases. On this point most authors, notably Forlanini, Brauer, Spengler, and Dumarest and Murard,[1] agree. The reasons are obvious. The fact that a case pursues a chronic course is an indication that the resisting powers are more or less good. In acutely progressing cases the local treatment of the lung does not overcome the low resistance, and tuberculous lesions of the other lung, or of extrathoracic organs appear, nullifying the reparative process in the treated lung.

Another group of cases in which artificial pneumothorax renders excellent service are those which have recurrent, copious, and uncontrollable hemorrhages. While, when afebrile, the patients are not in grave danger, and death due to exsanguination is rare, yet our efforts to prevent recurrence of hemorrhage after one has been stopped by keeping the patient in bed for several weeks are often futile, and he, as well as those around him, are discouraged. I have had some patients who had to remain in bed for two or three months with slight, but protracted hemorrhages, one following another. With the induction of a pneumothorax, provided we succeed in completely collapsing the lung, we have an excellent means of controlling the hemorrhage,

[1] La pratique du pneumothorax thérapeutique, Paris, 1919, p. 189.

to prevent its recurrence, and, in addition, to give the tuberculous focus an opportunity to heal.

Considering that the hemorrhage is stopped by the mechanical effect—by compressing the lung, and thus plugging the bleeding vessel, I used to fill the pleural cavity with gas during the first inflation; in one case I thus allowed 2000 cc of gas to enter. But further experience has taught me that such large quantities are not necessary. In some cases the injection of 300 to 500 cc of gas suffices to stop the bleeding, and I now am more conservative in this regard. On the next day several hundred cubic centimeters of gas are again permitted to enter the pleura, and refills are made according to indications.

It is obvious that only one lung may be compressed while the second must be left to carry on the functions of respiration, and that it is useless to combat a lesion in one lung while the disease is smouldering, or progressing in the other. For these reasons, it has been found advisable to apply pneumothorax only in unilateral cases. But as a matter of fact, in more or less advanced phthisis unilateral lesions are hardly, if ever, met with. Clinical experience is supported in this regard by autopsy findings. Inasmuch as strictly incipient cases are not to be treated by this method for reasons already stated, it is evident that in nearly all cases in which pneumothorax is indicated there will be found signs of involvement of both lungs and we must be satisfied with mild or moderate lesions in the untreated side.

In practice we find that in the vast majority of moderately and far advanced cases the lesions are extensive and active in one lung, while in the opposite there is limited involvement, or signs of quiescent or healed lesions. Though not strictly unilateral, these cases can be successfully treated by pneumothorax, if not prevented by pleural adhesions.

It is interesting that careful clinical and pathological observations have shown that only exceptionally is the untreated lung unfavorably affected. In spite of the increased functional activity because of the vicarious work it is compelled to do, the lung usually remains in the same condition as it was before the opposite lung was collapsed. The vicarious emphysema which is, as a rule, produced, increases its size, and dilates the alveoli and bronchioles, thus permitting as much air to be passed through as before, when both lungs were active. It is a common observation that active lesions in the untreated lung improve, or heal, after a pneumothorax is induced in the more affected side. The factors operative in such cases are not well understood. The increased blood supply may have something to do with it. The diminution in toxic absorption from the ulcerating and excavated lung may give the patient a chance to recoup his natural reparative forces, unhampered by the toxemia from extensive suppurating areas. However, this is not the rule. In some, lesions in the untreated lung flare up and extend, as has happened in some of my cases; copious hemoptysis even occurred from the untreated lung.

Forlanini, Brauer, Spengler, Saugman, Morelli,[1] and many others have argued that all advanced cases should be given an opportunity to benefit by artificial pneumothorax. In far-advanced, bilateral, or "hopeless" cases one side is, as a rule, extensively involved, while the other side shows only limited involvement, though the lesion may be evidently active. In such cases it is urged that the more affected side should be treated on the principle that there is nothing to lose and everything to gain. Forlanini's experience has taught him that when the untreated side has but a limited, even though active focus, the chances of success are better than would be expected *a priori*. When both sides are extensively affected the chances of recovery are slim indeed, but improvement in the general condition may be anticipated, and prolongation of life is not unlikely. At times, Forlanini says, we may be astonished that even such patients are cured. In most cases the removal or diminution of toxic absorption gives the patient an opportunity to muster his natural forces of resistance and comfort, often superior to that obtained in operative procedures for incurable cancer of the stomach, may be procured.

There is another important point to be borne in mind: We are not always able to ascertain positively whether the lesion in the less affected side is active, quiescent, or even healed. Rales and amphoric breath sounds heard over a given area of the chest wall are not always autochthonous, but may be in fact transmitted by conduction from the opposite side, and this is at times very difficult to differentiate, as was already mentioned. Indeed, perfect symmetry in location of rales, especially on both sides of the spine in the upper part of the chest posteriorly, should always excite suspicion that they may be transmitted, and on the side on which they are weaker it is probably so. The best procedure to ascertain such conditions is to auscultate from the affected to unaffected side by moving the stethoscope horizontally; if the rales remain the same in number and character, but more or less weaker, they are probably transmitted. We know that rales change in character in various lesions, and if we find the character and number of the rales on both sides remain the same throughout several days, it becomes more probable that they are transmitted from the sick side to the healthy side. With the collapse of the lung, the rales disappear, and those audible in the untreated lung may also disappear.

During and after pulmonary hemorrhage also there are often heard rales all over the chest which disappear in the unaffected side within several days, but when audible they give the impression that both lungs are extensively involved. Roentgenography is of little, if any, assistance in clearing up many of these cases.

Some French and Italian authors have suggested "diagnostic pneumothorax" in cases in which we are uncertain whether the disease

[1] Pneumothorax Artificial y otras intervenciones en la Tuberculosis, Montevideo, 1919.

is active in both sides. The more affected pleura is inflated with gas, and the opposite lung is watched. In cases in which the physical signs of disease are of the transmitted kind, they disappear soon after the lung is collapsed. But in case they persist in spite of a complete pneumothorax and the general condition of the patient is aggravated, the pneumothorax is allowed to be absorbed or, in more urgent cases, the gas is aspirated and the lung permitted to reëxpand. I have repeatedly resorted to this procedure and have, in rare instances, been rewarded by improving, or even arresting the progress of the disease in a case which appeared hopeless.

There are some who believe that even incipient cases ought to be treated with pneumothorax. Among these may be mentioned Murphy,[1] Lemke, Bullock and Twitchell, Gray,[2] Forlanini, von Adelung, Piéry, and some others. Murphy and Kreuscher say: "Is it well to wait until the outlook is so desolate? Is the lung collapse such a desperate operation as to be used only as a last resort?" With this I am not in agreement. If the treatment lasted only a certain and limited time, the patient could be informed of the details and given the choice. But, inasmuch as we are not in a position to give the patient definite information as to the probable duration of the treatment, and a large proportion of these cases recover under the old and tried methods, we should not subject mild incipient cases to the dangers, complications, and duration of pneumothorax. I still hold that only progressive or hopeless cases are to be given this treatment.

Contraindications.—To some extent the contraindications have already been given while speaking of the indications, but there remain yet to be discussed certain conditions which preclude the induction of an artificial pneumothorax, mainly those depending on the clinical form of the disease, the coexistence of extrathoracic tuberculosis, and of other diseases. Because pneumothorax only acts locally on the treated lung, acute miliary tuberculosis, in which both lungs are usually equally involved, is not suitable for this treatment. Fibroid phthisis with extensive pulmonary emphysema is not suitable for this mode of treatment, excepting when, in addition to the emphysema, there is a localized, suppurating excavation which is the cause of fever, sweats, cough, expectoration, etc., undermining the patient. An artificial pneumothorax may be applied as a palliative measure.

The most important forms of extrathoracic tuberculosis which complicate phthisis are laryngeal and intestinal involvement. Clinical experience has shown that pneumothorax may relieve these complications to an amazing extent. It appears that when the tuberculous toxemia, due to an active extensive focus in the lung, is removed by a pneumothorax, a slight laryngeal lesion often improves, and there are even some cases in which complete cure was obtained of

[1] Interstate Med. Jour., 1914, **21**, 266. [2] Illinois Med. Jour., 1913, **24**, 201.

both the lung condition and the extrathoracic lesions. A. de Gradi,[1] Zink,[2] von Adelung, and others, have reported such cases, and Forlanini speaks of them, though he confesses his inability to explain them. On the other hand, Rénon says that he was struck by the frequency of grave intestinal lesions found at the autopsies of cases treated by pneumothorax. Dumarest and Murard have observed frequently that mild intestinal lesions were aggravated soon after the induction of pneumothorax. This is in agreement with the writer's experience. Nearly all cases of pneumothorax that died under our observation had intestinal lesions. Conceding that the chances of cure are remote, slight laryngeal and intestinal complications should not deter us from applying pneumothorax if the case is otherwise suitable, on the principle that there is nothing to lose and everything to gain. Of course, advanced laryngeal lesions, with dysphagia, and intestinal ulceration, peritonitis, and amyloid degeneration of the viscera, are distinct contraindications to the induction of pneumothorax.

Diseases of the heart, bloodvessels, and kidneys have been found to materially lessen the chances of recovery with an artificial pneumothorax, and are therefore mentioned as contraindications to the treatment. They are all accompanied by disturbances of the circulation, and the patients do not bear the deprivation of the breathing area of a complete lung. Forlanini, however, has found that when compensation is good, pneumothorax may be induced with some chances of success. Some object to the production of a pneumothorax in persons over forty years of age. Rautenberg[3] reports death within two days after induction of pneumothorax in two patients over fifty years of age, and points out that rigidity of the thorax, pulmonary emphysema, etc., which go with advanced age, are distinct contraindications to this mode of treatment.

Diabetes has not been found to interfere with the successful outcome of an artificial pneumothorax, and the same is true of pregnancy. There have been reported several cases in which pneumothorax was induced in pregnant women who went on to term, were delivered of healthy infants, and continued under the treatment. In one of my cases the woman was six months pregnant when a pneumothorax was induced. The effect on the lung was excellent, complete collapse was attained and the general symptoms completely disappeared. The temperature chart (Fig. 127) shows clearly the effect on the fever, but she miscarried four weeks after the first inflation of gas. It is noteworthy that the temperature and the general condition of the patient were not influenced by the miscarriage.

Inasmuch as patients with artificial pneumothorax may have to go around with collapsed lungs for many years it is interesting that when affected with some disease requiring surgical intervention they may be

[1] Gazz. med. ital., 1910, **61**, 281.
[2] Beitr. z. Klinik d. Tuberkulose, 1913, **27**, 155.
[3] Ztschr. f. Tuberkulose, 1920, **32**, 1.

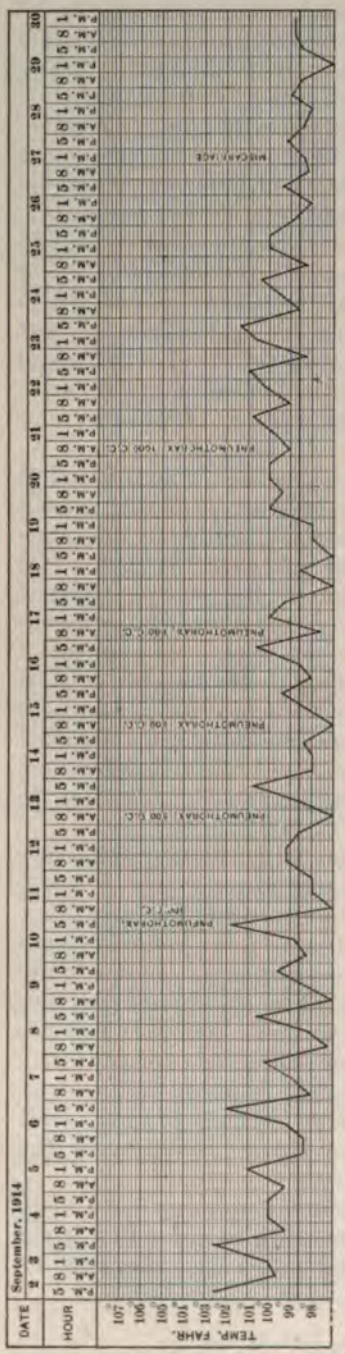

FIG. 127.—Effects of artificial pneumothorax on the temperature in a phthisical pregnant woman. The patient recovered.

given a general anesthetic. Reynier[1] subjected four patients to operations for tuberculosis of the kidney, intestine, mastoiditis, etc., giving them general anesthesia of chloroform and ether for as long as forty-five minutes. There was not observed any excessive dyspnea, cyanosis or shock during and after the anesthesia, the surgical wounds healed uneventfully, and the condition of the lungs and pleura remained unaffected by the surgical intervention.

Real[2] reports the case of a pregnant woman with a narrow pelvis and a small dermoid cyst. A Cesarean section was performed and the living child and the tumor were removed. Both the mother and the child did well.

Pleural Adhesions. — These are, strictly speaking, not necessarily contraindications to the induction of a pneumothorax, but they are hindrances to its successful accomplishment. In many cases no nitrogen at all can be introduced, because of extensive and dense adhesions and, after several punctures are made, the case is given up as unsuitable for treatment. Frequently an area is found which is free and some gas is introduced, but further attempts to introduce a sufficient quantity to completely collapse the lung meet with failure. This failure may be of various degrees. In some, the pleura is free only over a small area and a small pocket of gas can be made, while the rest of the pleura is adherent. No improvement in the condition can be

[1] Schweitz Rundschau f. Med., 1921, **21**, 277.

[2] Beitr. z. Klinik d. Tuberkulose, 1914, **29**, 349; 1916, **35**, 127.

expected and the treatment must be abandoned. Pleural adhesions often interfere with the treatment in a peculiar way. The pleura is free all over the chest, except its upper third, over the tuberculous lesion, where it is densely adherent. There may be a cavity in that location surrounded by stiff walls. The result is that, while we succeed in collapsing the lower two-thirds of the lung, the part which is diseased, and which we aim mainly at collapsing in order to expel the pus and detritus from the purulent cavity, and thus prevent toxic absorption and bring about coaptation of its wall with a view of giving them an opportunity to cicatrize, cannot be collapsed, and the disease keeps on its usual course. This is notably the case with old cavities having stiff fibrous walls which refuse to yield to the gas pressure. Many failures are due to this condition. Fig. 1, Plate XXII shows a roentgenogram of such a case. In spite of all efforts to collapse the lung completely, the adhesions around the lesion prevented the collapse of the diseased part of the lung.

Jacobaeus[1] has succeeded in cauterizing such adhesions and thus breaking them up permitting the lung to collapse. He uses for this purpose the thoracoscope, making but two punctures without producing a wide opening in the chest wall. Guided by the thoracoscope he locates the adhesions and cauterizes them with the glowing wire. His statistics of 37 cases show that in 27 he severed the adhesions so that complete collapse of the lung was obtained. From what I have seen of this procedure I have not been favorably impressed by it. It is not at all the harmless operation which its sponsor claims. A patient who is doing well should not be subjected to thoracoscopy.

At times the pleural adhesions are not very dense; in fact, slight adhesions are said to be present in practically all advanced cases of phthisis, and an increase in the pressure while introducing the gas breaks them up and success is finally attained—the lung is completely collapsed.

Proportion of Cases Suitable for Artificial Pneumothorax.—The proportion of cases suitable for the treatment is very small indeed. Statistics of most writers seem to indicate that less than 5 per cent of all cases that come under their observation are suitable for this treatment. Hardly 2 per cent of the cases that came under my observation during the past ten years could be considered suitable for pneumothorax treatment. Lemke[2] appears to be the only author whose clinical experience has been to the effect that he has had to abandon the operation in but a small proportion of the selected cases because of pleural adhesions. Perhaps the reason is that he operated on incipient cases. Bernard[3] found among 628 patients only 22 in whom he thought pneumothorax was indicated, and among these he succeeded only in

[1] Handb. d. Tuberkulose, 1914, **1**, 731; Surg. Gynec. and Obst., 1921, **32**, 493.

[2] Jour. Am. Med. Assn., 1899, **30**, 959, 1023, 1077.

[3] Le pneumothorax artificiel dans le traitement de la tuberculose pulmonaire, Paris, 1913.

6 cases in completely collapsing the lung, in 11 adhesions prevented the creation of a complete pneumothorax, and 3 refused to submit to the treatment. J. Courmont found among 352 patients only 31 that were suitable. Among 110 apparently suitable cases only in 32 per cent could Zink produce complete pulmonary collapse, and in 24 per cent he failed to enter the pleura altogether because of pleural adhesions.

Saugman found that in 33 per cent of the selected cases adhesions prevented the entry of gas into the pleural cavity. Even with Brauer's method, the proportion of failures exceeds 25 per cent. It must, however, be mentioned here that while in most cases complete collapse is best, a partial pneumothorax at times serves a good purpose, and many writers report excellent results when only creating one or more gas pockets in the pleura, and in some of my cases the improvement was remarkable under such conditions. Kuthy,[1] Faginoli, and, in this country, von Adelung even practise partial inflation of the two pleuræ *simultaneously* in bilateral cases, and they say that the results have thus far been apparently beneficial. To my mind this improvement can only be seen in chronic cases of phthisis, in which the cavities are surrounded by stiff walls of connective tissue, and which do not secrete any more. Exquisite amphoric breath sounds are heard over such cavities, but no rales. The excavations are not the cause of the constitutional symptoms which disable the patient, but the more acute patches of infiltration in other parts of the lung are responsible for the fever, nightsweats, etc. Compressing these parts we may achieve good results. In these cases we hardly ever achieve a cure with pneumothorax, because the cavity cannot cicatrize or contract owing to the stiffness of its walls which, together with the pleural adhesions, prevents its collapse by the gas pressure. But they may be greatly relieved by a pneumothorax. However, double pneumothorax is a very dangerous affair; I would not venture to induce it. We must always bear in mind the possibility of rupture of the lung, or of simple spontaneous pneumothorax, which may occur even when the most skilled operator is in attendance. In fact, several authors, especially Morgan, Faginoli, report rather unfavorable results with this procedure.

Duration of the Treatment.—The question how long the pneumothorax must be maintained in order to achieve a cure cannot be answered categorically; no rules can be laid down which will apply to all cases. In fact, considering that this method of treatment has been applied such a short time, there are few who have many cases under observation for from six to ten years, and even they have not agreed as to the usual duration of treatment of a successful case.

It appears that we cannot count on less than two years in the most favorable cases, although I have had success within one year in several cases the pneumothorax was allowed to be absorbed and there

[1] Centralb. f. d. Ges. Tuberkuloseforschung, 1921, **15**, 394.

occurred no relapse of the disease. But these cases are comparatively few. To my mind, the most difficult problem is to determine when the healing process has been completed, so that if the lung is permitted to reëxpand no active lesion will remain to flare up again by the respiratory movements. This, however, is difficult and, I believe, impossible to determine with any precision so long as there is complete collapse of the lung, and the general condition of the patient is good because of the collapse. Moreover, if we allow the pneumothorax to be absorbed too early there may not only be a relapse of the disease, but experience has shown that the pleural sheets are likely to adhere, and the fibrous bands prevent the formation of a new pneumothorax, if we find that this is indicated.

It is for this reason that whenever we decide to discontinue the treatment we must watch the patient carefully while the gas is slowly being absorbed, and if some symptoms appear, such as fever, cough, expectoration, anorexia, tachycardia, etc., we must at once reinflate the pleura. Forlanini says that many patients require a pneumothorax indefinitely, which is undoubtedly true, and most authors who have had experience with this method of treatment for many years, and had opportunities to observe their cases for long periods of time, agree with him.

In his most recent report of 172 cases observed for two to eleven years Saugman[1] says that he thinks "we may consider it a dangerous thing to cease treatment before the lapse of more than one year." He adds "in chronic cases I am much afraid of discontinuing the treatment before three or four years, and even after four years relapses are not impossible." He considers the cessation of a pneumothorax always, in a measure, a risk, and he lets the patient be treated for about five years. But Forlanini, Brauer, and myself have had in some cases good and even permanent results after six months' treatment. It is, however, better to continue for at least two years in all cases. In chronic cases we must consider two years as the absolutely shortest period of treatment, and in doubtful cases it must be prolonged for three and even four years. The inconvenience to the patient in having infrequent refills, four to six annually, is trifling considering that he can pursue his vocation, compared with the hazards of a relapse in case the lung is allowed to reëxpand too early. It is therefore better to continue the treatment for a year longer than to stop one month too early. If the disease is extensive it is advisable that the inflations should be continued over long periods of years, perhaps indefinitely.

Limitations of Pneumothorax Treatment.—We have shown in detail the remarkable results of artificial pneumothorax in the treatment of tuberculosis, and emphasized the fact that, unlike other modes of treatment, this has been applied in advanced and even hopeless cases, in cases in which the diagnosis is not in doubt, and which show a ten-

[1] Lancet, 1920, **2**, 685.

dency to progression. Because of these unquestionable merits some authors have pronounced it the most important advance in our attempts at curing tuberculous patients. But it has its limitations which should be mentioned before dismissing the subject.

To begin with, very few patients are suitable for the treatment; we have shown that the most sanguine find hardly 5 per cent of tuberculous patients can be classed as proper for the treatment, and I am inclined to agree with Rénon[1] that less than 1 per cent of all cases are really suitable. In other words, even if all the cases subjected to the operation were cured, which is not the case, as we shall soon show, 95 per cent of the sufferers from this disease would still have to be cared for by other methods of treatment.

The test of a cure should be made on acute and progressive cases and here pneumothorax shows that it is very often effective in reducing fever, removing symptoms of toxemia, etc. But we have seen that just in this class of cases the improvement is not permanent, as a rule. After an improvement lasting a few weeks or months there is, in most instances, a turn to the worse, the patient begins to lose out. In the chronic cases many brilliant results are attained. But the majority do well for a certain time until some complication compels us to abandon the treatment, and the patient must be cared for by the usual methods of treatment. In upward of 50 per cent of cases pleurisy complicates matters and does harm which is at times actually disheartening. In a large proportion, a lesion in the untreated side flares up and we must cease refillings. In still others, extrathoracic tuberculous lesions, especially of the intestines and larynx, complicate matters, and nullify the good effects attained in the treated lung. In a large proportion of cases we succeed in compressing the lung excepting the part which is the seat of the lesion, and which is held by dense adhesions which cannot be separated by gas pressure. In these cases we cannot claim success merely because they survived. Nearly all remain with symptoms of chronic phthisis. They survive without pneumothorax in many chronic cases.

Tuberculosis is a very costly disease; when treated with artificial pneumothorax it is even costlier. We have shown that the lung must be kept compressed at least two years, and in many cases more than five. Moreover, while many patients with collapsed lungs are well able to work, Rist[2] even reports the history of a patient with an artificial pneumothorax who withstood the hardships of war unscathed, the majority are practically invalids as long as they have a collapsed lung. The ability to do light work which many writers speak of in this connection is not unique to tuberculous patients with collapsed lungs. In every hospital for chronic and advanced tuberculous cases a large proportion of the patients are fit for light work, and there are

[1] Paris médical, 1921, **11**, 24.
[2] Presse Médicale, 1914, **22**, 692.

thousands of persons to be seen in cities who work more or less for years despite extensive quiescent lesions in the lungs.

It is clear that but few patients can raise the funds with which to support themselves for years while under treatment. It is decidedly wrong to induce a pneumothorax while the patient is in a sanatorium, and soon discharge him hoping that he will continue the treatment at home. In nearly all such cases that have come under my observation the treatment was abandoned soon after the patient left the institution. And it cannot be expected under present conditions that sanatoriums or hospitals should keep this class of cases for two, three, five, or more years. It appears to the writer that pneumothorax treatment can at present only be given to the rich or moderately well-to-do who are assured of skilful care for a number of years, and have patience to continue despite unfavorable complications.

Ultimate Results.—In the writer's experience the immediate results of the treatment are excellent; in some few cases brilliant. But the ultimate results leave much to be desired. It is to be regretted that of the thousands of cases that have been treated by this method during the past fifteen or twenty years, no properly collated statistics have been published as to the proportion of patients that have completely or partially recovered. The reason is clear when we consider that it is difficult, if not impossible, to compile statistics of results in a disease like tuberculosis which has such a variegated symptomatology, and course. The results will always depend on the number of acute and progressive cases that are involved, and the number of intensely chronic cases that may, and probably would, have done well without any treatment. If one institution should care for one or two hundred thousand cases, the statistical material would give us a fairly good picture of the results of the treatment. But when only one or two hundred cases comprise the number reported on, it is clear that half a dozen of very acute cases, or of very chronic cases, will change the result to an extent as to deceive. In fact, of the various reports of ultimate results published, hardly one is satisfactory to a trained statistician, and it is a notorious fact that hardly two authors have reported comparable material. Lemke hardly ever failed to induce a pneumothorax when others, equally competent, fail in 30 to 40 per cent of selected cases. The reason is, of course, that Lemke treated incipient cases, while others treat far advanced cases. Some limit their choice to strictly unilateral cases, others treat far advanced cases with lesions in both lungs; some avoid acutely progressive cases, others give them preference.

However, we may cite some statistical reports which give some idea as to the ultimate results of pneumothorax treatment. The most recent is that of Saugman,[1] who reports on 172 cases observed for from two to eleven years, and he compares them with 77 cases in which

[1] Lancet, 1920, **2**, 685.

he attempted to induce pneumothorax, but failed to collapse the lung because of adhesions, etc. He finds that of the 172 cases in which he succeeded in collapsing the lung, only 55, or 32 per cent, have recovered to a degree that "they are able to do general or light work;" 109, or 63.4 per cent, died from tuberculosis; 3, or 1.7 per cent, died from other causes; and 5, or 2.9 per cent, remained unable to work because of tuberculous disease. Bearing in mind that 16 of the 55 classed as able to work were still under treatment when the report was published, it is clear that the ultimate results in this series, while interesting, is not as brilliant as would appear on superficial examination. I have looked over my patients at the Montefiore Hospital from this viewpoint and found that of the cases with advanced tuberculosis necessitating hospitalization, 25 per cent could be classed as able to do "general or light work," and they have not been getting any special treatment.

A clearer picture of ultimate results in pneumothorax treatment may be gleaned from Dumarest and Murard's[1] recent book in which there are given figures about 113 cases observed for many years. Of these only in 8, or 7.55 per cent, has a complete cure been attained; in 7 improvement has occurred, but the lesions are still there, though quiescent; in 15, or 14.2 per cent, the lesions remained active, but less grave than before the treatment was instituted; in 26, or 24.5 per cent, the treatment was still continued when the report was published, but the lesions showed improvement; in 9, or 8.45 per cent, the treatment was still continued, but the improvement was not striking, and quiescent lesions in the untreated side existed; in 24, or 22.6 per cent, active and progressive lesions have developed in the untreated lungs; in 2, or 1.9 per cent, the treatment failed because of the poor general condition of the patients, and in 3, or 2.8 per cent, extrathoracic tuberculous lesions appeared; 8 patients, or 7.55 per cent, died as a result of pleural complications, empyema, perforation of the lung, etc.; and 4, or 3.8 per cent, died because of other complications.

From these figures it appears that an ideal cure, in which all the activity of the disease has been extinguished, the lung permitted to reëxpand, tubercle bacilli disappeared from the sputum and the patient became able to resume his occupation, was attained only in 8 out of 113 cases, or in 7.55 per cent. Of the rest, nothing striking has been shown. It must always be borne in mind when considering cures in tuberculosis, that a large proportion of active tuberculous cases survive for many years (see p. 410). Though the average duration of life of a person after acquiring tuberculosis is from seven to ten years, those with intensely chronic lesions live much longer. And it is in chronic cases that the success attained with this mode of treatment is most notable.

For these reasons it is not advisable to apply the treatment indis-

[1] La Pratique du pneumothorax therapeutique, Paris, 1919, pp. 151–159.

criminately, but that the cases should be selected with great care. I have felt that some cases in which I have applied this mode of treatment may have done better if they had been cared for in the usual manner. Indeed, after mature experience, I have arrived at the conclusion that a pneumothorax should be applied only in cases in which everything else has been tried but failed to give relief and there is nothing to lose by the procedure; that the potential pleurisies, pyopneumothorax, rupture of the lung, extrathoracic tuberculous lesions, etc., which nullify the good effects of pneumothorax in a large proportion of cases be all borne in mind, as well as the fact that collapsing the lung may prolong life, give comfort and even some efficiency which cannot be obtained by any other mode of treatment practised at present. Nearly all the present cancer surgery, of which many surgeons speak with justifiable pride, does not give results comparable with artificial pneumothorax in hopeless cases of pulmonary tuberculosis. No surgeon hesitates in performing the operation of gastrostomy for cancer of the esophagus or stomach, knowing that in all probability the patient will not survive three months. Palliative enterostomies, tracheotomies, etc., are performed with confidence that the best is done; even when life is not saved, comparative comfort is given during the last days of the unfortunate patient. Likewise, modern radiotherapy of cancer gives results which are considered good, the proportion of permanently cured is surely not higher than that obtained in tuberculosis with the aid of pneumothorax.

In hopeless cases of phthisis pneumothorax does more than the palliative surgery and radiotherapy in cancer: It removes or ameliorates the symptoms which make the life of the patient unbearable, the cough, the expectoration, the fever, the nightsweats, anorexia, hemoptysis, etc.; reinvigorates him and, in many cases, renders him efficient at his calling, or even to do some light manual labor, irrespective as to whether he is ultimately cured or not. The only inconvenience it puts him to is that he must report every month or two for a refill, which he knows from personal experience is painless, and bearable. In a small proportion of cases artificial pneumothorax is even more than palliative; it cures the disease radically and should therefore be applied in all cases where other methods of treatment have been tried but found wanting.

Other Surgical Operations for Phthisis.—Extrapleural Pneumolysis; Thoracoplasty.—Artificial pneumothorax is not the only method of surgical treatment of pulmonary tuberculosis. There have been suggested operations for the release of the compressed apex of the lung by the shortened first rib and ossified cartilage (p. 118); also injections of medication right into the lesion in the lung. Th. Tuffier,[1] in France, Sauerbruch,[2] C. Saugman[3] and, in this country, Willy Meyer,[4]

[1] Paris médicale, 1914, **4**, 231; Interstate Med. Jour., 1914, **21**, 259.
[2] Die Chirurgie der Brustorgane, Berlin, 1920, contains a thorough study of the subject.
[3] Tubercle, 1920, **1**, 305.
[4] Surg., Gynec. and Obstet., 1920, **31**, 161.

and Samuel Robinson have developed the operations of *extrapleural pneumolysis* and *thoracoplasty* with a view of compressing the affected area of the lung or complete collapse of the affected lung. The object of extrapleural pneumolysis is practically the same as that of artificial pneumothorax, but with this operation only the affected part of the lung is compressed while the rest of the parenchyma is left physiologically active. It can be applied in cases in which pneumothorax cannot, as when dense adhesions prevent the injection of air or nitrogen into the pleura.

A small piece of rib is resected over the tuberculous lesion, or the phthisical cavity which is surrounded by a thick fibrous wall, and an adherent pleura which prevents its shrinkage. The lung with both sheets of the pleura is then separated from the chest wall between the costal pleura and the endothoracic fascia. The lung is then collapsed so that the walls of the cavity are brought into apposition. The space thus created under the chest wall is filled in with Beck's bismuth paste, bismuth paraffin, or plain paraffin; Tuffier uses adipose tissue, fresh or preserved. The wound is then closed properly. No general anesthesia is used, because while squeezing out the secretions of the pulmonary cavity the lungs may be flooded, and aspiration pneumonia may be the result. But local anesthesia is sufficient according to those who practise the operation.

Tuffier urges his operation even in incipient cases, saying that we should not wait in phthisis till a cavity has formed, any more than we wait in tuberculous diseases of joints until suppuration or fistulæ have set in. But the modern treatment of tuberculous joint disease is rather conservative, and results are obtained which are superior to those obtained with operative treatment. It is doubtful whether the operation of extrapleural pneumolysis will ever become as popular as that of artificial pneumothorax.

Thoracoplasty.—Another method of attaining complete collapse of the tuberculous lung is the operation of rib mobilization which recently has been given preference by thoracic surgeons. To attain collapse of the chest wall the operation is usually performed in two stages. Some employ a general anesthetic, chloroform being preferred, while others use local, or regional anesthesia. A long incision is made on the posterior wall of the chest beginning above the first rib and extending downward along the outer border of the erector spinæ muscles as far down as the tenth or eleventh rib. Resection of the ribs is then done, beginning with the fourth, then the third, second and first, followed by removal of the fifth downward. Great skill is necessary to remove the second, and especially the first rib. The patient is then left for about eight or ten days, when the second stage of the operation is done, though in some cases, where the effects of the first operation are persistent, it may be necessary to wait several weeks before the patient is fit for further operative procedure. The aim is to remove the cartilages of the first to the seventh ribs.

Surgeons usually begin with the third rib and continue with the second and first, then begin again with the fifth and continue resecting the sixth, and seventh, while the fourth is left for the end, with a view of preventing great collapse before the end of the operation. The details of this operation are given in the papers by Meyer, Sauerbruch, and others, quoted above and especially in H. Morriston Davies'[1] book. While in the Tuffier operation only partial collapse is attained, rib resection gives complete collapse of the chest.

Phrenikotomie.—Another operation which has been suggested for the cure of phthisis is resection of the phrenic nerve with a view of procuring rest of the lower part of the lung by paralysis of the diaphragm on the affected side. F. Sauerbruch[2] and Stuertz have done this operation in Europe and Ralph C. Matson and Marr Bisaillon[3] have reported 2 cases in this country. It appears from the few cases reported that the operation is of no therapeutic value, if only because the diaphragm remains mobile with the respiratory movements after the operation.

More recently Warstat[4] achieved immobilization of the tuberculous lung by excision of the intercostal nerves. He argues that phrenikotomie only immobilizes the diaphragm and restricts the motion of the lower lobe of the lung, while the tuberculous process is almost invariably in the upper lobe. Cutting the nerve distal from the dorsal root from the second to the eleventh, inclusive, he succeeded in immobilizing the upper lobe of the lung. In animals he found that a few weeks or months after the operation the upper part of the lung was reduced in size and solid in consistency. In two patients in whom he thus operated he noted an unmistakable arrest of the disease.

These operations, and many more have been suggested and performed in isolated cases, may be attractive to the courageous surgeon, but they will appeal to the average medical man only in exceptional cases. Very few patients will submit to them. To be sure, they are recommended only in cases in which all other means have been tried, including therapeutic pneumothorax, but no relief has been obtained. But it is clear that strictly unilateral lesions are suitable; more so than is the case with artificial pneumothorax, which is rather innocuous when compared with thoracoplasty. The two successful cases seen by the writer have not swayed him in the direction of becoming an ardent advocate of surgical interference in pulmonary tuberculosis. The results in these cases could be called a surgical success: The patients survived the operation. But they remained sick with clinical tuberculosis of the lungs. Numerous patients go around for many years with such lesions, coughing, expectorating, and dyspneic without having incurred the dangers of this sort of surgery.

[1] Surgery of the Lung and Pleura, London, 1919, p. 212.
[2] München. med. Wchnschr., 1913, **60**, 625.
[3] Tr. Nat. Assn. Study and Prevent. Tuberc., 1915, **11**, 183.
[4] Deutsch. Ztschr. f. Chir., 1916, **138**, 437.

CHAPTER XLV.

GENERAL TREATMENT OF THE VARIOUS FORMS OF PULMONARY TUBERCULOSIS.

Incipient Phthisis.—The treatment of the early stage of phthisis, immediately after its recognition, varies with the intensity of the clinical manifestations of the disease. We have shown that a large proportion of cases manifest a strong tendency to spontaneous cure; the disease is "aborted" within a few months. These patients need no treatment beyond stopping work, keeping regular hours, increasing the quantity of food ingested, etc. A stay in the country for a month or two is even better. In most cases of this type institutional treatment is not advisable; in fact, I have seen some who were decidedly harmed by a stay in a sanatorium, where they were trained into carefully studying their disease, and impressed with the dangers of slight fever, fatigue, etc. Some have not been as industrious after the "cure" as before, though their state of health left little to be desired. With workmen having dependent families this is an important point.

It is different with patients in whom the disease manifests a tendency to acute progress; who have fever, nightsweats, cough, anorexia, emaciation, etc. These are to be given complete rest of mind and body until the acute symptoms are relieved. The best way of attaining this depends on the financial resources of the patient. The well-to-do may be treated at home, or sent to private sanatoriums. The results in either case will be the same in the vast majority of cases. Under no circumstances, however, should a patient with pyrexia be sent to the country, unless he can afford to take along a well-trained nurse, and will have competent medical advice. Febrile patients who cannot satisfy these two requirements are best treated at home, even if the home is only half-way satisfactory.

The principles of the rest cure, as well as of the treatment of pyrexia, have been given in detail elsewhere. Patients who cannot be managed at home along these lines should be sent to sanatoriums.

Patients with limited means should invariably be sent to institutions for the first few months of the disease, unless they can be moved into good homes where they may have appropriate rooms for themselves to carry out the rest and open-air treatment. But after remaining in the institutions for the period of pyrexia, they may return home where they may be cared for just as well as, and at less cost than, in the sanatoriums. Those who have no relatives or friends able and willing to give them a proper home should remain in the institutions

until the arrest of the disease is assured. As was already stated in Chapter XXXIX, the results are the same with home or institutional treatment, if the same amount of money is spent upon the patient in either case.

Reasonable patients, running only a subfebrile temperature, may be sent to the country for the first few months of the disease. Many improve to an astonishing degree, and are cured if the disease is of the milder or abortive variety. All patients should be sent out of town, preferably to the mountains, if there are no contraindications, for the hot summer months. During the winter most phthisical patients do well in the city.

The dietetics of phthisis have already been detailed in Chapter XL. But it should again be emphasized that patients with a good appetite and digestion need no special diet, except that they should eat more than they had been accustomed to before the onset of the disease. In many cases an increase in the quantity of proteins and fats is desirable. Those with anorexia and indigestion are to be treated for these conditions, because good gastro-intestinal functions are the best assets of the phthisical patient. A poor appetite, if not improved by open-air treatment, should be stimulated with some of the stomachic bitters; creosote in small doses is even better for this purpose in many cases. For indigestion appropriate dietetic and medicinal treatment is to be instituted.

In the vast majority of cases medicinal treatment is not necessary in incipient phthisis, unless it is for the relief of annoying symptoms. Cough may be controlled by the administration of creosote in moderate doses. In rare cases sedatives—codein, heroin, dionin, etc.—must be given in accordance with the indications discussed in Chapter XLIV. Anemia is to be treated with iron and arsenic. In fact, most patients treated at home should be given some medication, even if it is only a placebo, and for its psychic effect alone. But there is no doubt that ichthyol, creosote and arsenic, given intelligently, exert a good influence on the course of the disease.

The treatment of complications and special symptoms, such as hemoptysis, nightsweats, emaciation, etc., has been discussed elsewhere.

Most patients in the incipient stage of the disease do well under the mode of treatment just outlined. Many will recover within a few months; in a large proportion the disease will be arrested, but they are liable to suffer from relapses sooner or later. In many the disease will continue its onward march, irrespective of the treatment applied. We then have the so-called advanced stage.

Advanced Phthisis.—The zeal displayed by medical men during recent years to discover and treat early cases has resulted in neglect of those in whom the lesion has advanced beyond the stage which by common consent is called incipient. Hospital wards for advanced phthisis are often attended in a haphazard fashion, and the patients

are discouraged to a pitiable extent. Patients in the advanced stages are usually told by their medical advisers to go to some distant climatic resort, irrespective of their condition. This is all wrong. There is as much hope for the average patient in the moderately advanced stage as for a large proportion of incipient patients. Indeed, we have already emphasized the fact that the prognosis in advanced phthisis depends less on the age and extent of the lesion than on the acuteness and activity of the process.

A patient with an advanced, especially cavitary lesion, owing to the fact that he has survived the incipient stage, proves that he has a certain but variable amount of inherent resistance against the ravages of phthisis. It is our aim to preserve, or rather to increase, this power of resistance. This can only be done by proper regulation of diet, rest, and exercise, and by avoiding indiscretions which are liable to produce acute exacerbations of the tuberculous process.

We therefore regulate the diet of the patient in such a manner that he will not lack in assimilable nourishment (see p. 733). The question of rest and exercise is regulated under the guidance of the thermometer and the pulse-rate. In hopeful cases all efforts are to be directed at avoiding febrile exacerbations, or rendering them short-lived if they occur. Many of the afebrile patients may make themselves useful in some direction. Some may even work at their occupations, provided we find that they are not harmed by activity. The fact that one has cavities in his lung, or tubercle bacilli in the sputum does not mean that he is disabled. Patients engaged in vocations involving no undue muscular exertion may be very efficient. All should do something when strong enough to do it, but must cease all activities as soon as they feel fatigued, have fever, a rapid pulse, dyspnea, etc. This policy has during recent years been adopted in all the enlightened institutions for the care of the tuberculous, and the patients have benefited much more than by the previous routine rest treatment, carried out indiscriminately.

The diet in advanced phthisis is to be nutritious and of a character that will not overtax the digestive organs. At the least indication of indigestion, the diet should be appropriately corrected, because, next to fever, indigestion is most liable to hurt the patient irreparably. Those manifesting a tendency to obesity, and they are not so infrequent as is commonly believed, should restrict the ingestion of fats and carbohydrates. A fat consumptive is often more miserable than a lean one.

Medicinal Treatment.—The average patient is not satisfied with hygienic and dietetic treatment, and when no medicinal substances are administered he is apt to be led to the belief that there is no remedy for him. But there are drugs which have a beneficial influence on the course of the disease, as was shown elsewhere (Chapter XLI), and medication should be administered. Considering that the patient will have to be kept under control for months, it is often difficult to allay

his apprehensions and retain his confidence until the termination of the case. It is also a fact, to which we have already alluded, that while many remedies have an excellent influence on the disease or the patient, they retain their potency for but a short time, as a rule. The same is true of climatic resorts, and of institutions. The patients gain best during the first two or three months' treatment.

For these reasons medication must often be changed. Rénon's suggestion may be followed: The patient is given a course of several weeks with a certain remedy, and then it is changed for another medicament administered for several weeks. The results are often remarkable: There are gains in general health, the lesion in the lung shows signs of cicatrization, and the patient is encouraged. We may thus achieve the same results as with tuberculin without incurring the hazards of this dangerous preparation. A good method is to begin with ichthyol, administered as directed in Chapter XLI, for four or six weeks; or, if the patient thrives on it, it may be continued longer. For a week or two it is given in solution; for another fortnight in capsules, etc. Then we may give him creosote, or one of its derivatives —creosote or guaiacol carbonate, combined with arsenic, for several weeks. These substances may be given in mixtures, pills, globules, capsules, or by inhalation, as suggested by Beverley Robinson (see p. 752). Arsenic may be combined with creosote, or given alone in the form of Fowler's solution, or in pill form. Of course, if there is a tendency to hemoptysis neither the creosote nor the arsenic is to be given. The glycerophosphates are also beneficial, and may be given in appropriate doses. They exert an excellent influence on the tuberculous process, promote nutrition, improve the blood picture, etc.

Medication should be discontinued as soon as there is pyrexia, though when the temperature is below 100° F. medication may, and should, be given.

In addition to the above, there is to be given medication according to indications as revealed by the symptoms. The anorexia, night-sweats, constipation, diarrhea, etc., call for certain medicinal treatment which has already been discussed under symptomatic treatment.

In this manner the average tuberculous patient may get along very well for years. Some have very long periods of quiescence, and are only rarely laid up with acute exacerbations which need special treatment, as any acute condition. But they soon recuperate, as a rule, and again feel well for a variable period. While many survive acute exacerbations occurring at infrequent intervals, provided proper treatment is promptly instituted, in most of the chronic cases one of these acute exacerbations finally ends fatally. Many succumb to intercurrent diseases. These periods of quiescence may be obtained by judicious home treatment just as well as by institutional treatment, unless we are prepared to keep patients in sanatoriums for many years, irrespective of the activity of the disease. And acute and subacute exacerbations occur as frequently in sanatoriums and health resorts

as at home, provided, of course, that the patient is well cared for in his home.

Cases manifesting a tendency to progression, with acute or subacute symptoms and unilateral lesions, should be treated with artificial pneumothorax. It offers immediate relief of the symptoms, and shows more striking results than any other mode of treatment of active and progressive phthisis. Chronic but progressive cases are, at times, even more proper subjects for pneumothorax. The indications and contraindications are discussed in Chapter XLV.

Some cases show activity of the process despite the careful treatment. All efforts at raising the resisting forces are unavailing, and the disease progresses to a more or less speedy termination. All we can do is to apply symptomatic treatment, and to render the last weeks or days bearable and painless. *The solacing effects of the derivatives of opium should not be denied these unfortunates.* It is, however, one of the most common mistakes to send these patients to the country, or to sanatoriums. If the patient has a home in which there are no infants, he may remain there. If his financial resources are limited, the proper place is a hospital for consumptives. We are, at times, surprised that under proper care even the most desperate case recuperates, and within a few months returns greatly improved. Rarely, they even regain a capacity for working.

Treatment of Convalescent and Arrested Cases.—A large proportion of tuberculous patients in the advanced stages of the disease improve to an extent as to become useful at their respective occupations, although they have not been cured. They cough, expectorate, at times the sputum no longer contains any more tubercle bacilli, are more or less emaciated, but they have no fever, no tachycardia, etc. Physical exploration of the chest shows that there are cavities in the lungs, some displacements of the thoracic viscera, etc. Many of these are well able to take care of themselves, and even to be efficient at some easy occupation. Under proper medical supervision they may keep on in this condition for years, even for their natural lives. It is very important that these patients have some occupations, otherwise they are liable to brood over their condition and become actual hypochondriacs. The dependent ones are liable to intrench themselves in hospitals, and stay there indefinitely; when discharged, they soon seek admission to another one. They are very costly to the community, as well as to those depending on them. The fact that one has a cavity in the lungs or saprophytic tubercle bacilli in the sputum, does not mean that he is disabled from working any more than one who has a chronic tuberculous fistula or sinus in another part of the body. It is the intensity of the constitutional symptoms which should be the guide in these matters, and not the findings on physical exploration of the chest.

Once one has suffered from chronic phthisis of some duration, he is never cured in the anatomical sense; he is always in danger of

a relapse. He should be impressed with the fact that all that was attained was an arrest of the process, and that there may be at any time a recrudescence of the disease with even greater vigor than the former attack. These arrested cases should remain under medical supervision for several years, and examined periodically; first frequently, then at less frequent intervals, so that any tendency to a relapse may be checked early by proper treatment. While all efforts are to be directed toward prevention of excessive introspection and hypochondriasis, yet patients with arrested disease should be instructed as to the significance of certain symptoms, such as cough, fever, night-sweats, loss of weight, etc. During intercurrent diseases, especially catarrhal conditions of the upper respiratory passages and influenza, they are to drop all work and take a complete rest. In most cases these alleged intercurrent affections are really acute exacerbations of the tuberculous process of shorter or longer duration.

A patient with arrested disease should live in a healthy part of the city, in a good home, and sleep in a room with open windows. He may engage in his former occupation, excepting the dangerous ones, but the workshop must be of the modern and sanitary type, with good ventilation, etc. When possible, workmen should become gardeners, conductors, watchmen, chauffeurs, letter carriers, etc. When feasible, it is advisable that they take up farming. Well-to-do patients may move out of the city and settle for life in the country. Others may live in the suburbs, or in any country place where they can find suitable employment. Those who remain in the city should avoid indiscretions. But observing many of this class in the city, I feel that those in the country are, in the aggregate, not better off, and have no better expectation of life. The questions of marriage, pregnancy, and lactation have already been discussed.

Acute Phthisis.—The acute forms of phthisis are to be treated symptomatically, according to indications, so long as we have no specific for tuberculosis. In the pulmonary type of acute miliary tuberculosis careful hygienic and dietetic treatment is indicated. The nursing is of special importance, if we are to make the last days of the patient more or less comfortable. The treatment is the same as of any other acute or malignant infectious disease.

Acute pneumonic phthisis is not invariably fatal; often the patient passes the acute stage and becomes a chronic consumptive, and the treatment is then the same as that given above for chronic phthisis.

During the acute stage the patient is to be kept in bed, given food suitable for a febrile case, and the indications are otherwise met as they arise. When the acuteness of the process abates, the patient remaining with an active cavity, climatic treatment may be tried. Some of these patients do very well when removed from home to some place in the country, irrespective of its location or altitude. But they usually need a nurse or an attendant. The practice of sending such patients to shift for themselves in the country cannot be too severely censured.

It is unfortunate that public sanatoriums do not admit this class of cases.

Fibroid Phthisis.—The patient may feel well and be efficient at his occupation for many years, and the treatment at this period is purely symptomatic. It is, however, imperative to impress on him that overexertion and indiscretions are apt to activate the process.

Many patients with fibroid phthisis are well nourished during the latent or quiescent stage of the disease and need no special dietetic instructions. But we often meet with persons suffering from active or quiescent fibroid phthisis who are obese. The dyspnea, which is a marked symptom in this disease, is more severe in the fat consumptive, and it is advisable to arrange the diet so that the patient does not gain in weight excessively. Exceptionally, it is even necessary to reduce the amount of carbohydrates and fats with a view of reducing the weight of the patient. In my experience lean, even emaciated, individuals suffering from fibroid phthisis are more comfortable, and live longer, than those who are obese.

In many cases the iodides are very good. The dyspnea is very often relieved, expectoration is facilitated, and the general condition of the patient improves by the administration for several months of potassium iodide, or some of the newer albuminate compounds of iodine. But this remedy should not be given during febrile attacks, which are not frequent in this disease. When fever appears, and is persistent, the disease differs but little from common chronic phthisis. Those who are subject to hemoptysis, and many fibroid patients suffer from recurrent hemoptysis of varying severity, should not be given any iodides. It should be discontinued immediately at the appearance of streaky sputum. In many cases with profuse expectoration, creosote gives relief.

When signs of asystole make their appearance, with dyspnea, edema, etc., rigid rest in bed and appropriate doses of digitalis, strophanthus, etc., should be administered.

Fibroid patients should take frequent vacations. The mountains are not suitable for them because they are more short-winded the higher the altitude. It is best to send them to the plains or the sea coast. Many do very well indeed in a desert climate, provided they can adapt themselves to the surroundings, or "rough it."

In the later stages, when fever, nightsweats, cough, anorexia, etc., ensue, the case is one of advanced chronic phthisis, and is to be treated accordingly.

Pulmonary Tuberculosis in Children.—The acute types of tuberculosis in infants are hopeless, and the treatment is purely symptomatic. The infant is to be cared for as a case of pneumonia at that age. The only useful thing we can do for infants less than one year old is to prevent infection with tubercle bacilli. Once this has occurred, the prognosis is very unfavorable.

We have seen that chronic pulmonary tuberculosis of the type

common in adults is practically unknown among children under ten years of age. In them the disease manifests itself hematogenously, affecting the glands, bones, and joints, and is then the province of the surgeon, though it appears from all available data that hygienic and dietetic treatment have achieved better results than the knife in these cases. The physician encounters in children disease of the tracheobronchial glands. Considering that death due to this disease is very rare, it is clear that it is bearable by most children. The only problem is whether they are all destined to develop phthisis when reaching the age of adolescence, or later. This has not yet been solved to the satisfaction of all who are entitled to judge. We have seen (see p. 584) that when pulmonary tuberculosis does develop in one who had extrathoracic lesions during childhood, it is apt to pursue a very favorable course.

The treatment of tracheobronchial adenopathy aims at assisting Nature in its efforts to preserve the child. This can best be achieved by doing away, as far as possible, with the unnatural method of raising children. Growing children should not be kept indoors the greater part of the day and night, but should be urged to indulge in outdoor exercises and games. Especially is outdoor life imperative when a child shows signs of tuberculous infection, or of tracheobronchial adenopathy. These children should spend the greater part of the day outdoors, and sleep in rooms with open windows. If they can be raised in the country it is much better. But in every city, excepting the parts known as the "slums," children may enjoy outdoor life and benefit by it.

It must be borne in mind that children are easily adaptable to life in cold air, and most of them can run around the street with scanty clothing during very cold days and derive great benefit. They may also be given cold spongings followed by friction with a rough towel every morning, and thus "hardened." Only in this manner can "colds" be prevented in children. Harmless in themselves, colds may, in children with tuberculous glands in the chest, activate the tuberculous process and favor an acute exacerbation of the dormant tuberculous lesion. However, there are children who do not bear "hardening" well. If it is found that these cold spongings and rubbings are not followed by a proper reaction, or that they develop catarrhal conditions of the nose and throat, hardening should be given up. Many children are constitutionally unfit for cold baths, and they may be harmed by attempts to adapt them to these ablutions.

The ideal treatment of tuberculous children is to raise them all in the country. But like all ideals, it is only attainable by the favored few. The vast majority of infected children have to be raised in cities, for obvious reasons. But society, which is largely responsible for the conditions favoring tuberculous infection, can do a great deal toward saving these children, and raising them toward healthy manhood and womanhood, by providing vacations for them once or twice

annually, so that they may recuperate their vanishing forces and acquire resistance against the extension of the tuberculous process. In New York City this is done for a limited number of children derived from tuberculous stock by the Preventorium. In other cities in this country similar efforts have been made. But not all that need these vacations, proper food, and exercises are accommodated in any city.

If the parents of a child with tracheobronchial adenopathy can afford it they should move to the country, or to a suburb. In some cases it is feasible to send the child to be raised outside of the city lines. Many authorities maintain that it is best to raise these little patients in the mountains, or that they should be sent for frequent vacations to a high altitude. But I have seen excellent results in many cases which were sent to the seacoast, or to some forest climate. It is remarkable how quickly these children recuperate after a few weeks out in the open air, away from the city.

Many of these children do not eat enough, and the emaciation resulting from the smouldering tuberculous process in the chest is increased by the lack of nourishment. The anorexia is very often relieved by open-air life. A child in the city may not eat enough, or may have an actual abhorrence for food. But as soon as it is removed to the country, the desire for food is increased; often the appetite becomes ravenous a few days after arrival in the country.

In those who cannot afford to go to the country the anorexia may be relieved by open-air life in the city. They should be urged to spend the greater part of the day outdoors, and sleep in rooms with open windows. In urgent cases there should be no schooling. The modern open-air schools are of questionable utility, especially during the winter when the bitter cold is apt to prove unbearable to both the teachers and the pupils. The child needs not only fresh air, but exercise is just as important. This keeps the child warm in the coldest day. I have very little confidence in the educational value of the open-air classes; so far as I have observed, there is hardly any study during cold days. A child run down to such an extent as to need open-air life throughout the day and night is unfit for schooling, and should be taken out to the country for a few months or a season, or taken out of school for a similar period, until it recuperates, when it may resume studies.

The food of these children need not differ from that suitable for any child of the same age, but it should be plentiful, appetizing, and nourishing. It is even more difficult to place a child on a special diet than an adult. And there is no special need for such a procedure. It is, however, important to see to it that it does consume a sufficient quantity of proteins and fats. In children between two and four years of age, milk, cream, and eggs supply these requirements ideally. But older children should be urged to eat meats and poultry, and butter is the best source of fat for them. It is the most assimilable form of fat that can be given to the vast majority of children. Those who do not thrive on this diet, or who will not take a sufficient amount of

butter, should be given cod-liver oil. The vast majority of children take it pure, or with malt. Most of the emulsions contain very little of the oil, and are nauseous.

Children with enlarged bronchial glands will almost invariably do well under this mode of treatment. It is often astonishing to watch the recuperation of an emaciated child within one or two months after being placed under this treatment. It is encouraging to watch the great improvement shown by most of the children taken from the tenements of New York City to the country. In some obstinate cases it is necessary to repeat the vacation twice annually for several years. Some should be kept out of town until they reach adolescence. But it should always be remembered that they all do well if properly treated; the development of chronic phthisis before the age of ten is exceedingly rare, and infrequent before the age of fifteen.

There is, however, one danger to which these children are exposed. The endemic diseases of childhood, measles, whooping-cough, scarlet fever, etc., produce anergy or lowered reactive powers (see p. 127) to tuberculosis. They are therefore to be guarded against these diseases. Many a child, doing well despite tracheobronchial adenitis, succumbs to bronchopneumonia complicating measles or whooping-cough. It is very difficult to carry out prophylaxis against these endemic diseases in children living in the tenements of large cities; and in those who attend school in any part of a city, where there are so many "carriers." And we cannot isolate a child from intercourse with other children for obvious reasons. This is a fact which is often not considered in this connection by those eager to do something along these lines. If all efforts at prevention of complicating diseases have failed, and the child does develop one of them, the treatment should be very careful, and during convalescence the patient should be sent to the country for a month or more.

But infants can be shielded against infection with measles, whooping-cough, etc., because they are always in the immediate care of the mother. Infants known to have been infected with tuberculosis should be kept away from the proximity of other children who are liable to be "carriers." It is during infancy that measles and whooping-cough are likely to do most harm when attacking a subject harboring tuberculous infection.

Medical treatment is not indicated in most cases, excepting where there is anemia, cough, etc. These symptoms are best relieved by the open-air treatment. But we may in many cases assist or accelerate the improvement by the administration of iron. The old syrupus ferri iodidi may be given in doses of 3 to 5 drops to children three years of age, and more in proportion to older children. Iron tropon is another good and palatable preparation for these anemic children.

Children showing catarrhal symptoms, when not due to inflammatory conditions of the nose and throat, do well with creosote in small doses. It may be given in doses from $\frac{1}{4}$ to $\frac{1}{2}$ drop diluted in milk. Any of the derivatives of creosote may be given in powder or in syrup

form. This will often relieve a cough much more effectively than sedative drugs.

Specific treatment has been used with less success in children than in adults. It must be remembered that statistics of a number of children treated with any method, including tuberculin, are of no value if they show that of so many treated no deaths have occurred. Death due to tuberculosis, excepting meningitis, in children over two and under fourteen years of age, is exceedingly rare. For these reasons, orphan asylums show such splendid results—children of tuberculous parentage do not develop phthisis while they are in the institutions. But in children tuberculin is not indicated because the psychic effect, which is the main curative factor in adults, is lacking. I can see no reason for giving tuberculin to children.

Tuberculosis in the Aged.—Most aged phthisical patients are emaciated and debilitated. In many nourishment cannot be given in plentiful amount because they lack teeth for mastication, and most of them suffer from disturbances in the motility and secretions of the stomach and intestines. They also have arteriosclerosis, sclerotic kidneys, and do not bear the ingestion of large quantities of proteins. Fats are apt to induce diarrhea more often than in youthful subjects.

These difficulties in the dietetics of aged consumptives may be overcome within limits by first ordering the repair of the teeth. Then they may have a diet consisting mainly of milk, cream, and cereals. Fish is also well assimilated by aged persons, and they should take it when, for any reason, meats are not tolerated. But so long as the condition of the kidneys is not such as to contraindicate meats or poultry, they may be allowed in moderate quantities. Vegetables maly be given so long as there is no diarrhea. While in younger phthisical patients alcohol is to be tabooed, it is different with aged patients. If they have been accustomed to alcohol it is not advisable to attempt instituting reforms at an advanced age. In some cases alcohol is even of distinct benefit, if not abused.

Old patients do not bear outdoor life as well as younger ones. The same is true of high altitude. They must have warm rooms for living and sleeping. In fact, if they can afford it they should spend the winter in some southern region. The intense cold of the winter has a very deleterious effect on them because of the defective circulation—especially the peripheral—rigid arteries, sclerotic kidneys, pulmonary emphysema, etc., with which many are affected. But they need fresh air. While they should sleep in warm rooms, the windows must be kept open.

Cardiac derangements are to be carefully treated by rest, digitalis, strophanthus, etc. Myocarditis is, however, not relieved by these remedies and, in addition to rest, small doses of nitroglycerin, frequently repeated, often have a beneficial influence. The iodides are very good in many cases, and should be given in moderate doses. In many patients the dyspnea is relieved by this remedy much more effectively and lastingly than by anything else.

Fever is to be treated according to the principles discussed in Chapter XLIII. Most senile patients have no fever, but at times we encounter some with pyrexia of longer or shorter duration. Those in whom the fever is mild and evanescent require rest in bed until the temperature comes down to normal. Very old persons, over sixty years of age, do not bear fever very well, and must be given antipyretic treatment. Pyramidon in 5-grain doses may be administered three or four times a day.

The cough and expectoration need no treatment as long as they are not excessive. Otherwise, small doses of codein or heroin should be given. In many cases the expectoration is profuse and contains numerous tubercle bacilli. It may be greatly influenced by posture, as in bronchiectasis, and postural treatment may be attempted. But this is difficult with old persons, because of their weakness and debility they cannot withstand the vigorous cough this mode of treatment is apt to induce.

Tuberculosis during the Menopause.—Tuberculosis in women during the menopause is apt to be complicated by symptoms which are not seen in other phthisical patients. Considering the profound impression made by the tuberculous toxemia on the sexual sphere (see p. 610), there is no wonder that at the "critical period" tuberculous women should present special symptoms.

Many are more or less obese despite the continued activity of the tuberculous process in the lung. Dyspnea is very frequent, and many complain of cardiac palpitation. Hemoptysis is very frequent, and may replace the menstrual flow, though I should hesitate before considering it vicarious menstruation. Copious hemorrhages are uncommon; I am under the impression that they are less common than among others with similar lesions. But streaky sputum and small hemorrhages are very frequent. In addition there are most of the usual symptoms of the menopause—hot flushes, headaches, etc., and profuse perspiration. Combined with the symptoms of phthisis these symptoms of the menopause make this class of patients proper subjects for special treatment.

In addition to the treatment of phthisis outlined above, the special symptoms need attention. I have had several cases in which repeated hemoptysis was stopped by the administration of the extract of the ovaries or the corpus luteum. Indeed, most of the annoying symptoms which torture the unfortunate woman more than those caused by the tuberculous process, may be relieved by the timely and proper administration of these remedies. It is also a fact worthy of remembering that during the climacteric phthisical women do not bear the administration of tuberculin very well; most are apt to be harmed by specific treatment.

The cough and insomnia also are best relieved by the ovarian substance; sedatives and hypnotics often aggravate this condition, though in many cases bromides and valerianates are effective.

CHAPTER XLVI.

TREATMENT OF COMPLICATIONS.

Pleurisy.—Dry localized pleurisy occurring during the course of phthisis needs no special treatment, excepting to relieve the pain which is at times annoying. In mild cases external applications may suffice to give the patient comfort. Any of the belladonna plasters, or a sinapism may do; while some apply tincture of iodine. The writer finds, however, that the administration of salicylates often relieves these pleural pains much better than anything else. Aspirin, in doses of from 5 to 10 grains three or four times a day, may be given in cases in which sodium salicylate is liable to derange the stomach.

In acute cases of pleurisy the pain may be very severe during the first few days before the effusion appears and may necessitate the administration of morphine, $\frac{1}{8}$ to $\frac{1}{4}$ grain hypodermically. In most cases it is not necessary to repeat it, but it is better to strap the chest with adhesive plaster. In plethoric individuals, the application of several leeches to the painful area often relieves the pain when everything else has failed. As soon as the effusion appears the acute pain usually ceases. Cetrangolo[1] suggests the induction of an artificial pneumothorax for the relief of pain in acute pleurisy. He found that the insufflation of 100 cc of air into the pleural cavity increases the pain, but within twenty-four hours it disappears permanently. The cases in which this is indicated are rare.

The patient is to be kept in bed as long as the fever lasts. But during the later stages he may be permitted to take mild exercises. The diet is to be given in accordance with the temperature and the tuberculous process in the lungs.

It is not advisable to make any efforts to hasten absorption of the fluid in cases of tuberculosis. The fluid may be serving a useful purpose by compressing the lung and facilitating the healing of the lesion in the same manner as an artificial pneumothorax does, and also because of some biochemical effects (see p. 516). On this principle effusions may be permitted to remain for months. But in case the effusion causes cardiac embarrassment, severe dyspnea, cyanosis, insomnia, and other urgent symptoms, it should be aspirated at least partially. But even then aspiration should be left as a last resort, because speedy withdrawal of the fluid, and rapid expansion of the lung may awaken the tuberculous process in the parenchyma into

[1] Semana Med., 1919, **26**, 161.

acute activity. The writer has observed this to happen in several cases.

It is best to first try autoserotherapy. Five to 10 cc of the fluid are withdrawn with an aspirating syringe and reinjected into the subcutaneous tissue. A good way is not to remove the needle after the syringe is filled with the fluid, but while withdrawing it, when its point reaches the subcutaneous tissue, to turn it parallel to the surface of the chest and to inject the fluid right then and there, as was described by the writer[1] elsewhere. This can be done several times on alternate days. In most cases there will be noted an increase in diuresis, and the level of the fluid begins to sink, so that within a couple of weeks it may be absorbed altogether.

In cases in which autoserotherapy is of no avail, and the general condition of the patient demands removal of the effusion, aspiration should be done. It is advisable not to remove all the fluid at one sitting, but to do it on alternate days, each time withdrawing a part. In many cases the pleura refills soon after tapping, and it is necessary to assist the absorption by giving a salt-free diet, and to reduce the amount of fluid ingested by the patient. Diuretin may be of assistance by increasing diuresis. But other drugs, reputed as assisting absorption of pleural effusions, as the iodides, are impotent in this regard. Emptying the bowels daily with salines, if there are no contraindications, may assist in the absorption of the fluid.

Many authors treat pleurisies by withdrawing the exudate and replacing it with air, claiming that in this manner reaccumulation of fluid is prevented. But we have seen that while troublesome, the presence of fluid in the chest is not dangerous *per se*. It is only the tuberculous process in the parenchyma of the lung that is dangerous. And this is as well controlled by the fluid as by air in the pleural cavity. Moreover, converting the pleurisy into a pneumothorax does not prevent the reaccumulation of fluid, as experience has taught the writer. This is natural when we consider the frequency of exudates in pneumothorax. It is also to be borne in mind that a hydropneumothorax may become purulent, and then the prognosis is very serious.

Empyema.—The treatment of purulent effusions, during the course of phthisis is very unsatisfactory. Some authors have stated that when the pus shows diplococci, especially pneumococci, and also streptococci and staphylococci, the prognosis is better, and resection of one or two ribs may bring about a cure, while in cases in which the pus shows the presence of tubercle bacilli, operation is futile. In the experience of the writer there has been observed no difference from this viewpoint. In very rare instances we meet with a case in which several aspirations of the pus cure the empyema. Similarly the writer has had cases of localized and encapsulated empyemata which broke through bronchi, the pus was expectorated and the patients recovered. In

[1] Jour. Am. Med. Assn., 1913, **60**, 962.

the vast majority of cases we keep on withdrawing larger or smaller quantities of pus, but the chest fills up again in a short time. In some cases fistulæ form along the track of the needle, discharging pus externally.

The results of operations for empyema complicating phthisis are unsatisfactory. A simple incision for the evacuation of the pus is nearly always followed by a fistula necessitating the patient to go around with a foul-smelling bandage for the rest of his life. For this reason most physicians are at present satisfied with the aspiration of the pus, repeated according to indications. Recently I have seen excellent results with the Carrel-Dakin method of treatment, and now urge it in all cases of pyothorax complicating tuberculosis.

Whether treated by aspiration or thoracocentesis, the fever usually keeps up, dropping after the removal of part of the pus, but rising again within a few days. Emaciation, nightsweats, anorexia, diarrhea, etc., keep on; amyloid degeneration of the viscera, notably the liver, spleen, kidneys, and intestines, develops and the patient sooner or later succumbs to exhaustion.

The suggestion of some authors that after removing the pus nitrogen should be inflated into the pleura has been tried by the writer, not found to offer any advantages, and abandoned.

Pneumothorax.—Considering the morbid anatomy of pneumothorax complicating pulmonary tuberculosis, it is clear that the prognosis is vere serious. Bearing in mind that the rent in the visceral pleura occurs in tuberculous tissue, its chances of cicatrization are remote. However, if the rent is small, intrapleural pressure and collapse of the lung, at times, help along in the direction of healing of the laceration, and we then may even have what some writers have called "providential pneumothorax;" the collapse of the lung results in a temporary or even permanent improvement in the general and local condition of the patient. But when the rent in the pleura is large, it remains open indefinitely and the morbid secretions of the lung lesion enter the pleural cavity through a pleuropulmonary fistula and infect it.

The acute onset with shock, pain, dyspnea, etc., demands active treatment. The indications are clear: The patient is to be relieved of the urgent and menacing symptoms, his heart is to be stimulated, etc., which is best done by a hypodermic injection of morphine. But if the patient is not calmed, and the dyspnea is urgent, thoracocentesis is to be performed. This is often the only means at our command to relieve the extreme and agonizing dyspnea. Tapping the air in the affected pleural cavity gives prompt relief, though unfortunately only of short duration in most cases. Plunging a hypodermic needle into the affected side is sufficient, because the expiratory pressure within the pleura is greater than that of the external atmosphere. It is good to attach a rubber tube to the needle by one end, while the other is placed in a pail of water, thus forming a water valve which permits the free exit of the air from the chest, but prevents its return.

If the relief thus obtained is only transitory, the operation is repeated; in some cases it may be necessary to repeat the tapping four, five, or even seven times during the first day. Some have tried to obviate this by inserting a cannula and leaving it in the chest wall for several hours or days; the rubber tube all the time in the water. But I have found it very difficult to retain the needle in place and to keep it aseptic. For this reason I prefer to make several punctures as the urgency of the symptoms demand.

Many theoretical objections have been raised against tapping the chest in these cases. But one has only to witness a case in which the agonizing pain and air hunger are promptly relieved by tapping, to appreciate that this is the only measure which gives relief. As in urgent cases of any kind, theoretical considerations are left until the menacing symptoms are under control. In fact, after one tapping the patient begs for another when the dyspnea returns.

I have recently been more successful with induction of counter-pressure within the pleura by injection of air in the way we do when inducing a therapeutic pneumothorax. This was first suggested by Morelli. It appears from actual measurements that in the vast majority of these cases the intrapleural pressure is negative, even though the patient suffers from severe dyspnea. Increasing the amount of air in the pleura the perforation is closed by the air pressure, the edges are held together and they soon heal. Closure of the fistula prevents further entry of septic matter from the lung into the pleura. As done by A. Pisani,[1] a needle connected with a manometer is introduced into the pleura and, if the pressure is found positive, some of the air is withdrawn. Then the tube leading from the needle is connected with the usual pneumothorax apparatus and air is allowed to enter the pleural cavity until the manometer registers 5, 10, or even 20 cm. positive water-pressure.

In several cases in which this method was tried by the writer, relief was noted immediately in two out of three. In some cases we may continue the pneumothorax treatment, just as we do in cases of therapeutic pneumothorax.

We meet with cases in which the embarrassment of the circulation and respiration continues in spite of repeated tappings, or introduction of air, and the prognosis is gloomy. The causes are not primarily mechanical, but physiological. The opposite lung is congested and the circulation is thereby more embarrassed than by the displacement of the mediastinum. In these cases we may try oxygen inhalation, and cupping all over the posterior aspect of the chest. Some use wet cups or venesection to relieve the right ventricle which is becoming paralyzed from extreme overdistention. "I have no doubt," says West, "that life might be sometimes saved by timely venesection, and it is certain that bleeding is not so much employed in these urgent cases as it ought to be."

[1] Gazzetta degli Ospadali e delle Cliniceho, 1917, **37**, 379.

The heart action is to be sustained by large doses of digitalis, spartein or camphor.

In milder cases, especially those in which the pneumothorax is only partial and the symptoms are not so urgent, the treatment is less vigorous. The dyspnea, pain, and distress are usually controlled by a dose of morphine hypodermically, and within a day or two the patient feels quite comfortable. Partial pneumothorax is very frequent in tuberculous patients, and commonly is followed by improvement in the tuberculous lesion in the lung.

The after-treatment, if the patient survives three or four days, is that of the underlying tuberculous process in the lungs. Inasmuch as the pneumothorax, with its sudden onset and agonizing symptoms, often leaves the patient in a debilitated condition, rest and proper feeding are to be enforced. In rare cases the pneumothorax, acute and menacing as it was at the onset, turns out to be "providential," as some French authors say. The collapsed lung is given an opportunity to heal and recovery may take place ultimately. Some recommend that in such cases the pneumothorax should be continued by injections of nitrogen in the approved manner.

After the menacing symptoms have abated, the patient, regaining his strength and composure, provided he has no fever, may be permitted to leave his bed and take mild walking exercises. We know now from experience with artificial pneumothorax that one can do considerable exercise, or even work, while one pleural cavity is filled with air and the lung collapsed.

Hydropneumothorax.—The treatment of effusion into a pleural cavity filled with air is conservative, just as that of pneumothorax. The fluid is absorbed sooner or later spontaneously. We now have experience with this condition in cases with artificial pneumothorax. So long as there is no fever or dyspnea, the patient may be allowed considerable exercise. But in case the intrathoracic pressure becomes high and produces dyspnea when the patient is at rest, the pressure must be reduced. This can be done by withdrawing some of the air or fluid. The latter is the best. With an aspirating apparatus a part of the exudate is withdrawn. In many cases the operation has to be repeated. In favorable cases this withdrawal stimulates the absorption of the rest of the fluid. In several cases I have had good results with autoserotherapy (p. 857).

Pyopneumothorax.—The treatment of this complication is very unsatisfactory. Operative interference has not given encouraging results. At best, a fistula is left in the chest which discharges pus indefinitely. The ultimate result is worse than when only tapping of the pus is resorted to. The indications, therefore, are to aspirate the pus at frequent intervals with a view of keeping the patient afebrile as far as possible. The bacteriological findings have no influence on the prognosis and treatment, as has already been stated when speaking of empyema complicating phthisis.

Laryngeal Tuberculosis.—Many cases of tuberculous laryngitis show a strong tendency to spontaneous cure, especially in patients whose lung lesion also manifests a tendency to improvement. In fact, the progress of the lesion in the larynx goes hand-in-hand with the progress of the lung lesion, though the physical signs of the latter are apt to be obscured by the former. This is clearly seen in cases in which the induction of a therapeutic pneumothorax is effective in curing the patient. If there has been a laryngeal lesion it often shares in the general improvement of the patient.

In my experience, local treatment is not often effective in enhancing cicatrization of laryngeal lesions. When carried out vigorously, it is apt to do harm. The application of local escharotics and cauterization has been harmful in the long run, or of no benefit in the vast majority of my cases. As has been pointed out by St. Clair Thomson,[1] lactic acid, which is the favorite drug used by laryngologists, is unavailing except in strength of 50 per cent or more. Hence, sprays of 2 per cent are nothing but irritating. Frequent applications are also irrational, the object being to produce an eschar which does not separate for one or three weeks. When the slough is detached a healing ulcer is exposed; but there are generally deeper deposits, requiring a repetition of the cauterizing process, so that four to twelve applications may have to be spread over as many months. The use of a 20 to 25 per cent solution of argyol, or a 2 per cent solution of methylene blue for local application, is less likely to be painful or harmful. Where the mucous membrane is unbroken no local application of drugs does any good.

The futility of local applications of any kind in superficial tuberculous lesions is clearly seen in tuberculous lesions of the skin. Dermatologists are not so sure of promoting a healing process by the application of escharotics to lesions of lupus, as are laryngologists in tuberculosis of the larynx. I have often felt that my patients had better chances of recovery before local treatment of laryngeal lesions was applied than after vigorous treatment.

In a few cases I have seen excellent results when the patient ceased talking altogether, thus affording perfect rest to the larynx. But it must be done thoroughly. The patient should have a pad and pencil and carry on all conversation in writing. In the cases, mostly women, in whom this treatment was carried out perfectly, the laryngeal lesion healed. There are, however, few patients who have sufficient will power and perseverance to continue this treatment for a long time. In patients with advanced and active lesions in the lungs, there is no reason for trying it, because they are doomed anyway.

As has been shown by Fetterolf,[2] there is one form of the disease in which unlimited use of the voice is advisable, this being the variety in which the vocal cords are the only parts of the larynx involved.

[1] Diseases of the Nose and Throat, New York, 1912, p. 606.
[2] Hare's Modern Treatment, Philadelphia, 1911, **2**, 402.

This is commonly called the "chorditic" form, the cords appearing slightly congested and having on their upper, and to a slight extent on their mesial aspect a number of reddish granular growths. These are possibly sometimes submucous tubercles, but more frequently are distended mucous glands with their duct orifices occluded. Vocal exercise aids in clearing up the condition, and it is in this form that improvement of the voice so frequently follows an acute coryza.

In all cases with dysphagia palliative treatment must be applied. We may try to obtain relief by laryngeal insufflations of 3 to 5 grains of orthoform or anesthesin. It is only effective when there is ulceration and the powder remains on the ulcer. If given about one hour before the main meal the patient may be comfortable for a whole day. The following formulæ may also be used:

℞—Orthoformi	gr. xxx	2.0
Iodoformi	gr. xxx	2.0
Mentholi	gr. vj	0.4

M. S.—Insufflate a few grains one hour before meals.

℞—Cocaine hydrochloridi	gr. x	0.7
Morphinæ hydrochloridi	gr. ij	0.1
Mentholis	gr. xv	1.0
Iodoformi	ʒij	8.0
Acidi borici	ʒij	8.0

M. S.—Insufflate a few grains one hour before meals.

The application of these powders is to be made with special insufflators. They are designed so that the spray goes vertically downward, not backward into the pharynx.

In some cases the dysphagia is severe and not at all influenced by the application of remedies locally. Injections of alcohol into the superior laryngeal nerve may then be tried. Relief from pain may be obtained lasting several weeks. Rudolf Hoffmann was the first to suggest this mode of treatment. The technic of the injection is thus given by J. Dundas Grant:[1]

Place the patient in a horizontal position and, with the thumb of the left hand, press the sound side of the larynx toward the middle line so that the affected half projects distinctly; the other fingers of the hand lie on this. The index finger enters the space between the thyroid cartilage and the hyoid bone from without until the patient announces that a painful spot had been reached. With a little practice one arrives at it at the first go-off, when one has become familiar with the topographical relations. Now the nail of the index finger is placed on the skin (which has been previously disinfected) in such a way that the point of entrance for the needle lies opposite its middle. The needle is pushed in for about 1.5 cm. and this distance is marked off on the needle perpendicular to the surface of the body. According to the thickness of the subcutaneous layer of fat, the

[1] Lancet, 1910, 1, 1754.

perforation has to be more or less deep. The needle is then carefully moved so as to seek a spot at which the patient states that he feels pain in the ear. The syringe filled with 85 per cent alcohol warmed to

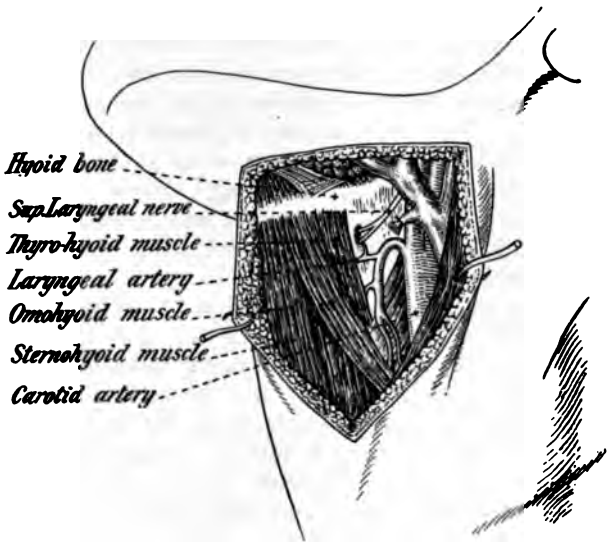

FIG. 128.—The thyrohyoid region. (Grivot.)

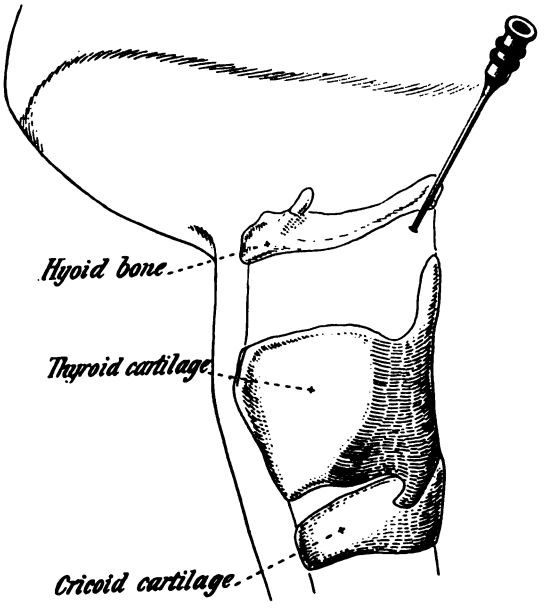

FIG. 129.—Space where to insert the needle for producing anesthesia of the superior laryngeal nerve. (Celles.)

the temperature of 45° C. (113° F.) is screwed on to the handle and the piston is then slowly pressed down. The patient now feels pain in the ear, the passing off of which he indicates by raising his hand. During the operation he has to avoid both swallowing and speaking; if, however, he makes a movement of swallowing we must follow the movement of the syringe with a light touch. The injection is kept up until no further pain occurs in the ear; then the needle is removed and collodion or adhesive plaster is placed on the spot of the injection without pressure. The needle employed should be one with a point bevelled off much more obtusely than in an ordinary hypodermic needle, so as to avoid the risk of puncturing a vessel.

I have tried this method in many cases and obtained relief for the patients in about 50 per cent. Failures are due to missing the nerve, which is unavoidable in many cases.

There are cases in which all the above fail to relieve the sufferer, and all we can do is to give large doses of anodyne drugs. In some we may obtain relief by helping the patient in the following manner while he eats: A trained person stands behind the patient and makes firm and even pressure at the angle of each jaw at the moment of swallowing. Another way is known as Wolfenden's position: The patient lies prone over the bed with the face over the end and sucks the nourishment through a glass tube from a cup held by an attendant or placed on the floor. These maneuvers seem cumbersome, to say the least, but when having under our care a patient who cannot swallow even water without severe pains in the throat, we are ready to try anything.

There remains yet to mention the various operations of curettage and cautery which laryngologists perform in these cases. Some employ direct laryngoscopy while operating, but this is not only violent, but the results have been disastrous in all the cases that have been done for me. In advising operation to a patient of this class we must first ascertain the general and the local condition of the lungs. In case the prognosis is poor because of the general condition, there is no reason for operating. I always object to operations in febrile and cachectic patients.

INDEX OF AUTHORS.

A

Abderhalden, 116
Adami, 63, 107
von Adelung, 800
Albrecht, 107
Aldrich, 508
Alexander, 394, 553
Allard, 513, 518
Allbutt, 487, 727
Als, 824
Amenomiya, 565
Ameuille, 509
Amrein, 295
Amstel, 580
Ancell, 62, 257
Anders, 234, 241
Anderson, John F., 57, 207, 285
Andral, 122, 240, 339, 598
André, 45
Andrews, 108
Antylus, 755
Aretæus, 241, 302
Arkin, 754
Arloing, 41
Arluck, 44, 451
Armand-Dellile, 662
Arneth, 283
Arnould, 621
Arnsperger, 363
Ascarelli, 638
Aschoff, 169
Ash, 534
Assmann, 357, 363
Atwater, 740
Auche, 108
Aufrecht, 53, 126, 149, 325
Ayer, 175

B

Babes, 45
Baccelli, 617
Bach, 522
Bacmeister, 50, 53, 118, 121, 139, 391
Balboni, 807, 813
Baldwin, 24, 38, 111, 114, 668, 763
Ballenger, 559
Balzer, 555
Bamberger, 287
Bandelier, 344, 389, 415, 766
Bang, 110, 780
Barbier, 286, 314
Bard, 415, 419, 524
Bardswell, 216, 276, 621, 724, 745, 767
Barjon, 496
Barnes, 754
Barot, 467
Barr, 113, 115
Bartel, 50, 115, 143, 156, 455, 583, 610 716
Barth, 589
Barthez, 594
Bartlett, 57, 63
Bartlett, J. R., 150
Barwell, 558, 561
Fateman, 741
Bauer, 114, 123, 610
Baumann, 140
Baumgarten, 53, 106, 153, 161, 163
Bayle, 368, 419, 512, 558
Beale, 223
Beck, 390
Béclére, 565
Beddoe, 299
Behrend, 387
Behring, 59, 142, 662, 770
Beitzke, 41, 52, 55
Bell, 525
Benda, 437, 568
Benedict, 698
Bennett, 257
Bergel, 715
Bergheim, 112
Bernard, 44, 835
Bernard, Claude, 349
Bernheim, 662
Bertillon, 81
Besredka, 392
Bezançon, 314, 415
Biach, 523
Bialokur, 609, 612
Bibb, 362
Bickersteth, 794
Biermer, 405
Biggs, 81, 692
Binet, 112
Birch-Hirschfeld, 52, 69, 109
Bisaillon, 843
Bisbee, 648
Bittorf, 521
Black, 272
Blake, 462

Blakiston, 514
Bloomfield, 598
Blum, 287
Blumberg, 523
Blumenfeld, 282
Boardman, 206, 362
Boas, 290, 556, 593, 596
Bodington, 696
Bohland, 280, 592
Bollog, 584
Bonney, 314, 562, 787
Borel, 74
Börschke, 566
Boston, 383, 414
Bowditch, 512
Bowlby, 471
Bramwell, 514
Brandenburg, 609
Brauer, 602, 795, 798, 800, 819, 822
Bray, 214, 215, 220, 340, 343, 349
Brehm, 284
Brehmer, 111, 231
Brieger, 260
Broders, 614
Bronfenbrenner, 392, 511
Brooks, 662
Brown, 24, 47, 265, 411, 598, 766, 769
Brown, E., 474, 477
Brown, Wm. G., 669
Brownlee, 42, 59, 98, 129 134, 154
Brown-Séquard, 249
Bruce, 420, 680
Brückner, 387
Brunon, 149
Brunton, 724
Budd, 257
Bullock, 832
Bulstrode, 128
Burnand, 63, 68, 226
Burns, 23, 242, 265
Bushnell, 34, 97, 127, 158, 189, 284, 339, 346, 352, 664, 668
Busse, 598

C

Cabot, 243, 246, 351
Calmette, 47, 55, 56, 73, 144, 389, 392, 666
Calvin, 786
Capps, 293, 483, 484
Carr, 132, 455
Carrel, 826
Carrington, 682
Carroll, 394
Carson, 794
Castaigne, 524
Castellani, 551
Cattermole, 71
Cavagnis, 107
Celles, 863
Cepulic, 394
Cetrangalo, 856
Chalier, 109
Chalmers, 552
Chamaro, 602
Chamberland, 108
Chambers, 62
Chantemesse, 480, 511, 569
Chapin, 728
Chapman, 216, 734, 745
Charvot, 574
Chauvet, 379
Chiari, 555
Childs, 363
Chittenden, 739
Claisse, 612
Clark, Andrew, 257, 371, 419, 421, 580
Clark, James, 257
Claypole, 257
Clemenger, 210
Clough, 285
Clouston, 294
Clovis, 598
Cobb, 118
Cobbett, 34, 41, 56, 68, 130, 140, 142, 158, 685
Cochrane, 769
Cohen, M. Solis, 267, 290, 556
Cohen, S. Solis, 114, 604, 608
Cohn, 361, 362
Collis, 133, 134, 135, 863
Combe, 294, 449, 739
Condie, 234
Coonley, 663
Coriveaud, 514
Cornet, 23, 46, 68, 133
Cornil, 290, 589
Corper, 135, 140, 255, 272, 276, 754
Cotton, 22, 56
Councilman, 57
Courcoux, 480, 511
Courmont, 45, 836
Couston, 513
Cowan, 530
Cowie, 109
Craig, 282, 392, 393
Cramer, 136
Crofton, 112
Cruice, 564, 573
Cruveilhier, 589
Cullen, 579
Cummer, 532
Cummings, 697, 700, 701
Cummins, 74
Cursham, 569
Curtin, 757
Czerny, 592
Czyhlar, 592

D

Da Costa, J. C., 465
Da Costa, J. M., 324
Dakin, 826
Damman, 42
Daremberg, 220, 434, 743, 477
Darier, 269, 270
Dastre, 785

Davies, 843
Davis, 27
Debains, 392, 629
Debove, 734
Dehn, 363
Deibel, 580
Delafield, 174
Deléarde, 144
Delépine, 23
Delhern, 189, 535
Dellile, Armade, 000
Delpeuch, 299
Demme, 663
Demoiseau, 491
Deneke, 44
De Renzi, 754
D'Espine, 466
Destrée, 290
Dettweiler, 218, 737, 772
De Witt, 749, 750
Dieulafoy, 245
Dioscorides, 754
Doane, 22
Dobell, 257
Dobrovici, 657
D'Oelsnitz, 290
Dold, 45, 284
Donaldson, 495
Dorset, 21
Dowdell, 569
Doyen, 355
Drasche, 523
Drummond, 739
Duboff, 683
Dubrull, 513
Duckworth, 599
Dumarest, 829, 833, 840
Dunham, 362, 493
Duplay, 574
Dürck, 50
Dworetzky, 558, 559
Dzierzgowski, 116

E

Eastwood, 19
Eden, 57
Ehrlich, 510, 747
Eich, 580
Einhorn, 260
Elderton, 150, 624, 647
Eliasberg, 395, 586
Ellis, 491
Emerson, 524
Emerson, Haven, 588
Engel, 294
Engelmann, 48
Ernst, 44
Escherich, 455
Estor, 638
Etiénne, 569
Evans, 295
Ewart, 174, 314, 351, 409, 465, 574
Ewing, 588

F

Faginoli, 836
Faisans, 279, 612
Farrago, 395
Felner, 580
Fenwick, 199, 257, 260
Fenwick, W. S., 257
Féré, 122
Fernet, 510
Fetterolf, 322, 683
Fildes, 392
Findel, 140
Finkler, 539
Fisac, 133
Fischer, 285
Fischera, 551
Fisher, 740
Fleiner, 479
Fleischner, 663
Flexner, 550
Flick, 784
Flicker, 479
Fleissinger, 716
Flint, 254, 323, 512, 514, 618
Floresco, 785
Florschütz, 648
Floyd, 481, 818
Flügge, 46, 48, 49, 698
Fochi, 784
Folin, 739
Fontana-Tribeudeau, 552
Foot, 163
Forchammer, 584
Fordyce, 157
Forget, 598
Forlanini, 795, 797, 804, 829, 837
Forssner, 581, 583
Forster, 584
Forsyth, 759
Fowler, 171, 555, 615, 763, 766
Fox, 195, 234, 253
Fraenkel, 770
Fraenkel, A., 535
Fraenkel, C., 45, 140, 295
François-Frank, 249
Franz, 390
Fraser, 40
Fraser, Thompson, 154, 711
Freudenthal, 561
Freund, 118, 139, 580
Frey, 785
Freymuth, 149
Frias, 70
Friedmann, 29, 31, 106, 657
Friedrich, 405
Frieseke, 535
Frischauer, 553
Fulton, 97
Funk, 234, 257, 261, 286, 534
Fussell, 522

G

Gabb, 522
Gabbet, 19

Gaffky, 57
Galen, 255, 302, 755
Ganghofner, 387
Garb, 733
Gardner, 141, 153
Garland, 491
Garnier, 663
Garrahan, 70
Gärtner, 106
Garvin, 539
Gassmann, 261
Gaube, 112
Geddes, 124
Geipe, 108
Geisbock, 281
Gerhardt, 250, 535
German, 486
Ghon, 57, 108, 168, 450
Gibson, 468
Gibson, Cole B., 394
Giesmann, 804
Gilbert, 140, 283, 410, 614
Gillet, 539
Gilliland, 362
Gilmore, 272
Gimbert, 578
Giraux, 638
Glaister, 83
Glaser, 392
Glover, 392
Goebel, 50
Goethe, 253
Goetz, 613
Goldscheider, 314, 331, 336
Goldtwait, 123
Goodale, 143
Gordon, 48
Goring, 150
Gornault, 153
Goujot, 574
da Gradi, 883
Graetz, 210
Graham, 525
Gramen, 395
Grancher 257, 314, 337, 465, 789
Grant, 126
Grasser, 44, 639
Grassmann, 44
Grau, 638, 639
Graves, 608
Grawitz, 282, 479
Gray, 526, 570, 832
Greene, 643
Greenfield, 207, 608, 609
Gregg, 140
Griffith, A. S., 20, 33, 34, 40, 64, 68
Griffith, F., 64
Grivot, 863
Grober, 479
Grocco, 602, 493
Grysez, 56
Guerin, 144, 668
Gutstein, 812
Guyenet, 564
Gye, 136

H

Hahn, 638
Halbron, 313
Haldane, 698
Hall, D. C., 703
Hall, F. De Haviland, 244
Halter, 133
Halverson, 112
Hamburger, 608
Hamburger, F., 37, 49, 57, 76, 121, 388, 472
Hamman, 390, 391, 513, 522, 539
Hammer, 392
Hanau, 581
Hanford, 611
Hanot, 589, 614
Hans, 539
Hansemann, 138, 368, 765
Hansen, 110
Harbitz, 51, 63, 64, 143, 443
Harras, 118, 121
Harrington, 296, 530
Harris, 223
Hart, 69, 118, 121, 139
Hartley, 566, 570
Haupt, 150
Haushalter, 569
Haven, 283
Hawes, 351, 558, 640
Hayek, 621
Hayem, 260
Head, 289
Head, Henry, 292
Heberden, 274
Hedenburg, 748
Hedges, 513
Hefflebower, 629
Hein, 231
Heise, 24
Hellin, 526
Helmers, 754
Hempelmann, 451
Henderson, 286
Henderson, John T., 573
Henke, 181
Herard, 589
Hermann, 205
Herter, 739
Hess, 49, 58
Heublein, 469
Hierokles, 569
Hilderbrand, 107
Hill, Leonard, 698
Hillenberg, 72
Hinsdale, 715, 720
Hippocrates, 61, 123, 299, 502, 580
Hirsch, 76
Hirtz, 570
His, 120
Hofbauer, 553
Hoffman, 74, 77, 78, 80, 91, 132, 133
Holeman, 123
Holst, 359
Holt, 44

Honeij, 467
Honl, 109
Honsele, 638
Hoppe-Seyler, 477
Horetzky, 638
Howell, 467
Hrdlicka, 73
Huber, 296
Humphrey, 571
Hunter, 569
Hutchinson, 257
Hutchinson, Woods, 73

I

Igerscheimer, 32
Inman, 392, 694
Iscovesco, 759
Iselin, 574
Iwai, 123

J

Jaccound, 632
Jackh, 107
Jackson, 551
Jacob, 72
Jacobaeus, 835
Jacobson, 296
Jacoby, 210
Jadassohn, 271, 584
Jakowski, 510
von Jaksch, 211
James, 51
James, T. L., 716
Jani, 107
Janowski, 258
Jeannil, 638
Jeannin, 268
Jelliffe, 295
Jessen, 392, 617, 820
Jex-Blake, 243, 542
Jochmann, 602
Jones, 51
Jordan, 359
Joseph, 210
Jupille, 392, 629

K

Kaminer, 580
Keats, 786
Kehl, 608
Keith, 122
Kellogg, 738
Kelly, 484
Kelynack, 614, 615
Kenchington, 645
Kennerknecht, 284
Kernig, 441
Kessel, 276, 285, 391
Kettle, 136
Keysser, 126
Kidd, Percy, 514, 558, 599
Kienböck, 530
Kiewe, 580
Kindberg, 189, 511, 535
King, 736
King, J. T., 351
Kinghorn, 626
Kitassato, 58
Kjerrulf, 111
Klebs, 33
Klemperer, 152, 260, 822
Klenke, 24
Klimmer, 764
Kline, 563
Klipstein, 50
Klotz, 186, 329
Knight, 718
Knipfelmacher, 451
Koch, Herbert, 451, 453
Koch, Max, 29
Koch, Robert, 17, 29, 46, 95, 145
Kocher, 608
Kohler, 579, 705
Kohlisch, 46
König, 638
Köniğer, 515
Koplik, 470
Koranyi, 465
Koslow, 284
Kossel, 58
Köster, 513, 518
Kraus, F., 582
Krause, Allen K., 24, 145, 361, 363, 447, 579, 683
Kreuscher, 832
Kreutzfuchs, 359
Krönig, 314, 327, 329, 537
Krumwiede, 28, 33
Kruse, 29
Kuban, 132
Kupferle, 580
Kurashige, 284
Küss, 168, 392, 747
Kuthy, 30, 111, 201, 237, 411

L

Laennec, 62, 144, 241, 368, 396, 512, 618
Lafitte, 114
Laignel-Lavastine, 114
Lamallerée, 29
Lancereaux, 614
Landis, 581, 583, 615
Landouzy, 299, 510
Lange, 30, 32, 564
Langstroth, 292
Lanz, 394
Lasbennes, 580
Laschtchenko, 48
Latham, 105, 724
Lauritz, 261
Leaming, 346
Lebert, 564, 580

Lee, 698
Lees, 314, 379
Lehmann, 108
Lemgey, 638
Lemke, 832
Lémoine, 603
Lenglet, 584
Leplat, 674
Leredde, 392
Lésague, 258, 571
Lesne, 569
Lesieur, 45
Létulle, 172, 173, 297, 522, 578, 612
Leube, 729
Leudet, 411
Levaditi, 157
Levene, 36
Levison, 257
Levy, 150, 156, 281
Levy, F., 313
Lewandowsky, 45, 271, 584, 585
Lewis, 741, 749
Leyden, 568
Libman, 249
Lichtheim, 249
Liebermeister 284, 569
Limbeck, 282
Lindhagen, 89
Locke, 276
Loeffler, 207
Lombard, 62
Lombardi, 301
Lombroso, 122
Londe, 109
Longa, 510
Longet, 249
Longstrath, 292
Loomis, 371
Lord, 253
Lorentz, 614
Lorrain, 539
Louis, 195, 232, 234, 253, 523, 564, 569, 598
Löwenstein, 29, 42
Lubarsch, 52, 53, 63, 108, 134, 614
Lundborg, 123
Lunde, 786
Luschka, 317
Lustgarten, 30
Lyon, 816

M

McCann, 113
McCarthy, 253
McCartney, 794
McCaskey, 615
McConkey, 143
McCrae, 63, 171, 534
McIntosh, 392, 393
McLean, 692
McNeil, 154
McSweeney, 724, 731
Mace, 483
Macfadyen, 134
Macht, 21, 247, 784, 785
Mackenzie, James, 483
Mackenzie, Morel, 558
Mackenzie, Hector W. G., 765
MacWhinnie, 702
Maffucci, 23, 25, 106
Magnus-Alsleben, 238
Magnus-Levy, 605
Mahler, 22, 112, 257, 261
Mallory, 57
Mandl, 793
Mann, 593
Mannheimer, 786
Manning, 71
Manoukhine, 392, 630
Mantoux, 70, 229
Manwaring, 511
Maragliano, 600, 770
Marcellus-Empricus, 755
Marcet, 284
Marcus, 41
Marfan, 260, 663
Marie, 275, 716
Marmoreck, 770
Marquard, 579
Martius, 155
Massol, 392
Mathieu, 657
Matson, 843
Mauthner, 115, 611
Maximow, 163
Mayer, 152
Mayo, 152
Mays, 618
Meader, 20
Means, 813
Melchior, 261, 613
Meltzer, 813
Mendel, 739, 741
Meriel, 565
Metchnikoff, 73, 674, 739
Mettetal, 39, 391
Metzger, 629
Meyer, A., 210, 530
Meyer, K. F., 663
Meyer, Willy, 841
Microli, 617
Milchner, 106
Milian, 600
Miller, H. R., 392, 630
Miller, J. L., 539, 775
Miller, William Snow, 479
Mills, 286, 573, 598
Mioche, 70
Mitchell, 34, 154
Mitchell, Weir, 796
Moeller, 111, 112, 195, 253
Mohr, 126
Möller, B., 32, 49, 153
Moncanny, 114
Mongour, 150, 569
Mönkenberg, 69
Montaugh, 393
Montgomery, 276, 286, 287, 580, 605

Moore, 78, 217
Morelli, 831, 859
Morgan, 801, 818
Morgenroth, 151
Morin, 114, 608
Moritz, 134, 800
Morland, 221, 766
Moro, 44, 71, 388
Morris, 493
Mortley, 210
Morton, 197, 263, 602
Mosenthal, 573
Mosiman, 608
Mowat, 360
Much, 20, 41, 153
Mudd, 126
Müller, B., 241
Müller, F., 50
Müller, Hans, 783
Munoyerro, 70
Münstermann, 566
von Muralt, 114, 291, 589, 716
Murard, 829, 833, 840
Murphy, 295, 808, 832
Müsemeier, 42
Musser, 304
de Mussy, 280, 483

N

Naegeli, 63, 68
Nakari, 107
Nattan-Larrier, 172, 173
Naunyn, 605
Neelsen, 19
Nehring, 82
Neisser, 46
Netter, 510
Newman, 116, 121, 247
Newsholme, 128, 132, 729
Nikolski, 521, 522
Nocard, 25
Nolf, 551
Norris, 332, 580, 581, 583
Nothnagel, 235
Novack, 108

O

Oestreich, 323
O'Farrell, 252
Ogle, 91
Oliver, 110
Opie, 66, 68, 414, 521
Ormsby, 267, 268
Orth, 41, 57, 156, 642
Osborne, 739
Osler, 513, 553, 554, 724
Otis, 189
Ottenberg, 249
Overland, 72

P

Page, 496
Paget, 584
Paillard, 197
Palmer, 580
Pankow, 580
Papavoine, 62
Papillon, 289
Pappenheim, 29
Parfitt, 626, 727
Park, 18, 22, 26, 27, 28, 33, 40
Parr, 580
Parrot, 168
Pasquera, 46, 48
Paterson, 511, 693
Patzold, 44
Pawlow, 741
Pearce, 57
Pearl, 96, 104
Pearson, 62, 93, 96, 104, 105, 110, 150 728
Pehu, 109
Pensunti, 510
Penzold, 220, 678
Peretz, 351
Peron, 774
Perry, 647
Peter, 197, 199
Peters, L. S., 564, 602
Peters, W. H., 578
Petersen, 555
Peterson, 482
Petri, 27, 30
Petroff, 21, 24, 47
Petruschky, 150, 391, 600
Pettit, 592, 601
Pfannenstiel, 30
Philip, R. N., 328
Philip, W., 257
Philippi, 766
Pidoux, 598, 603
Pierce, 513
Piéry, 233, 269, 314, 420, 624, 800, 832
Pietrzikowski, 638
Piller, 89
von Pirquet, 71, 128
Pisani, 859
Plesch, 320, 406
Plummer, 609
Polanski, 122
Pollack, 257, 603
Pollak, 70
Poncet, 602
Pope, 150, 411
Porter, 103
Porter, A. S., 116
Porter, William, 780
Potain, 287
Pottenger, 122, 266, 307, 313
Poujade, 690
Powell, 281, 371, 495, 523, 525
Power, D'Arcy, 599
Pradal, 313
Preissich, 391
Price, 246
Prudden, 174
Prudella, 580

Q

Querner, 285
Quryat, 449

R

Rabinowitsch, 29, 30, 41, 69, 144, 156, 391, 764
Radcliffe, 392, 601
Radziejewski, 387
Ramond, 490
Randt, 395
Ransome, 91, 93, 126, 128
Raphael, 207
Rasmussen, 176, 253
Rautenberg, 833
Ravenel, 52, 53, 54, 57, 285
Raw, 23, 604
Real, 834
Regnault, 592
Regner, 834
Reibmeyr, 298
Reiche, 237, 241, 580, 602
Reichenbach, 749
Reinecke, 395
Reinhart, 64, 68
Reinhart-Goodwin, 255
Rénon, 114, 656, 747, 834
Reuben, 451, 453
Reuschel, 387
Revault, 569
Ribbert, 53, 139
Richet, 738
Riddell, 530
Riesman, 522, 539
Riess, 408
Rietschel, 108
Rilliet, 594
Rindfleisch, 118
Ringer, 785
Risel, 63
Rist, 189, 511, 535, 667, 800, 802, 838
Ritter, 153
Rivers, 123
Riviere, 154, 314, 336, 340, 379, 766
Rivolta, 25
Robin, 112, 286, 729
Robinson, Beverley, 752
Robinson, Samuel, 574, 842
Roger, 44, 48
Rogue, 290
Rokitansky, 116, 580
Roland, 510
Rolleston, 614
Rolly, 388
Romanowski, 552
Römer, 43, 54, 60, 109, 145
Rondot, 756
Röpke, 344, 289, 415, 766
Rosalino, 123
Rosenau, 58
Rosenberg, 716
Rosenberger, 143, 284
Rosenfeld, 82
Rosignol, 145
Rossalimo, 123
Rossel, 824
Rosthorn, 580
Rousseau, 253
Roux, 25
Rubel, 687
Rubinstein, 392
Ruedinger, 410
Ruge, 569
Rumpf, 770
Ruscher, 645
Russell, 112

S

Saathof, 609
Sabourin, 221, 229, 556, 580
Sabrazes, 569
Sahli, 763
St. Aude, 291
St. George, 614
Sainton, 313
Sajet, 89
Sajous, 114
Sale, 522
Salters, 233
Sampson, 365, 565
Sander, 40
Sands, 616
Sauerbruch, 826, 841, 843
Saugman, 144, 227, 798, 800, 836, 839
Sawyer, 465
Saxe, 295
Saxtorph, 793
Sazuki, 284
Schade, 581, 583
Schäffer, 581, 583
Schaffle, 265
Schauta, 580
Scheel, 63
Scheppelmann, 522
Scherer, 611
Schern, 284
Schick, 451
Schiff, 395
Schlachter, 104
Schlimpert, 577
Schlossberger, 31, 32
Schlossmann, 81
Schlütter, 04
Schmidt, 39
Schmorl, 52, 108
Schröder, 22, 56, 653, 720, 767
Schule, 522
Schulze, 121
Schwatt, 276
Schweitzer, 519
Scott, 573
Sears, 512
Sedlmayr, 804
Selter, 82, 140, 156, 210
Senator, 287
Serbonnes, 398

Sergent, 291, 338, 353, 381, 486, 556, 599, 612
Sewall, 314, 381, 713
Shingu, 796
Shortle, 787
Sieber, 116
Simmonds, 107
Simon, 211
Singer, 337
Siredey, 612
Sitzenfrey, 198
Sluka, 464
Smith, 121
Smith, Eustace, 451, 467
Smith, F. C., 719
Smith, Theobald, 21, 23, 25, 40, 53, 101
Sokolowski, 234, 420, 428, 592
Sommerfeld, 133
Soparkar, 23
Sorel, 756
Sorgo, 234, 241, 253, 274, 388, 602, 770
Soulignoux, 574
Spano, 107
Spehl, 551
Spengler, 795, 818, 822
Spieler, 455
Spindler-Engelsen, 207
Spivak, 791
Sprawson, 769
Squire, 475, 626, 784
Stadler, 726
Staehelin, 526
Staines, 716
Steffenhagen, 42
Stern, F., 562
Stern, R., 638
Sticker, 126
Stierlin, 565
Stiller, 122
Stimson, 392
Stivelman, 824, 825
Stockwell, 550
Stoerck, 344
Stokes, John H., 585
Stokes, William, 271, 312, 794
Stoll, 512
Stoll, H. F., 462, 467, 469, 512
Stone, 613
Strandgaard, 241, 265
Strauss, 51
Stricker, 243, 251, 770
Strohl, 800
Sukienikow, 462, 463
Sweet, 77
Sydenham, 580
Symmers, 588

T

Tachau, 802
Takaki, 284
Takeya, 45
Taute, 29
Taylor, 638, 729
Tecklenberg, 580
Tecon, 22
Tendeloo, 117, 178
Tenzer, 388
Tesler, 580
Thayer, 493
Thaysen, 140
Theodorescu, 313
Thilenius, 802
Thiroloix, 612
Thoeni, 140
Thom, 150
Thompson, 241, 624, 724, 767
Thompson, E. H., 692
Thompson, J. C., 361
Thompson, St. Clair, 562
Thormeyer, 567
Thue, 253, 510
Tibber, 455
Tibbles, 743
Tobiesen, 802
Todd, 257
Tonelle, 564
Torrey, 186
Townsend, 580
Toyofuko, 146
Traube, 231
Tripier, 568
Trousseau, 569, 784
Trudeau, 618, 696, 723
Tuffier, 841
Turban, 111
Turk, 739
Twitchell, 832

U

Uhlenbrock, 144
Uhlenhut, 206
Ullom, 282
Ungermann, 57
Urban, 642

V

Vandervelde, 221
Vaquez, 569
Vastenburgh, 56
Vaughan, 600
Vehling, 153
Verneuil, 574
Villar, 574
Villemin, 17, 24, 25, 284, 598
Viole, 551
Virchow, 770
Vischer, 522
Vitvitski, 522
Vogeler, 684
Volk, 145
Von den Velden, 783
Voss, 112

W

Wagner, 47
Wallace, 785

Wallgren, 586, 818
Walsh, 361, 362, 364, 564
Walshe, 234, 523
Walther, 612
Wang, 586, 666, 786
Ware, 243
Warnekross, 115
Warren, 89, 136
Warren, E., 580
Warstat, 701
Warthin, 108, 109
Washburn, 22
Watson, 629
Webb, 114, 150, 283, 410, 682, 687, 716, 802, 818
Weber, 29
Weber, E. Parkes, 638, 642, 664
Weber, H., 150
Weichselbaum, 51, 143, 284
Weigert, 161, 165
Weil, 496, 523
Weinberg, 104, 661
Weiss, 484
Weisz, 629
Weisz, M., 116
Welch, 221
Weller, 109
Wells, 748, 758
Wenckenbach, 121, 530
West, 243, 313, 522, 524, 599
Wetherhill, 626
Weygandt, 578
Wheaton, 266
White, 95, 287, 539, 762
Whitla, 55
Whitney, 521
Whitney, H. B., 78, 152
Widal, 495, 569
Wiedersheim, 119
Wiese, 221
Wilcox, 94
Wildbolz, 393, 394
Williams, C. Th., 148, 234, 253, 523, 618, 758
Williams, F. H., 359
Williams, Mary E., 459
Williams, R., 615
Williams, W., 586
Williamson, 82, 605
Wilner, 638
Wilson, 247
Wincourof, 44, 451
Winsch, 253
Winslow, 698
Wintrich, 404
Wittgenstein, 116
Wolfenden, 864
Wolff, 253
Wolff-Eisner, 37, 201, 322, 411, 817
Wollstein, 51, 63, 109
Wolman, 362, 364, 390, 391, 539
Wood, 55
Wood, J. W., 361
Wood, N. K., 319
Woodruff, 129
Wright, A., 210, 283
Wright, L. B., 600, 757
Wunderlich, 603
Wynn, 217, 459

X

Xylander, 206

Y

Yeld, 614
Yeo, 150
Ysendick, 580

Z

Zahn, 522
Zemmin, 824
Ziegler, 361
Ziehl, 19
Ziemann, 73
Zink, 833, 836

INDEX OF SUBJECTS.

A

Abortion in phthisical women, 675
Abortive tuberculosis, 414
  diagnosis of, 417
  physical signs of, 417
  symptomatology of, 415
  treatment of, 845
    climatic, 717
Abscess, cold, of chest wall, 574
  ischiorectal, 182, 613
  of lung, 546
Acid-fast bacilli, 18, 29, 30
  in blood, 284
  in milk, 57
  streptothrix, 550
  in tap water, 383
Acnitis, 268
Actinomycosis of lung, 549
Activity of disease, determination of, 190
Acute forms of tuberculosis, 424
  prognosis in, 426, 620
  treatment of, 849
  miliary tuberculosis, 436
    course of, 446
    diagnosis of, 444
    meningeal form of, 441
      in adults, 442
      in children, 441
    pathogenesis of, 436
    pathology of, 438
    prognosis in, 446
    pulmonary form of, 439
    roentgenology of, 445
    traumatic, 642
    treatment of, 849
    typhoid form of, 439
    tuberculin test in, 387
  pneumonic phthisis, 430
Addison's disease, 610
Adenoids and tuberculosis, 588
Adenopathy, bovine bacilli in, 33
  cervical, 457
  tracheobronchial, 457
    in children, 457
    diagnosis of, 470
    pathology of, 168, 184
    physical signs of, 461
    prognosis in, 471
    roentgenology in, 468
    symptoms of, 457
    treatment of, 850
    tuberculin diagnosis in, 470
Adrenals, dysfunction of, 609
  in etiology, 114
Age incidence, 83, 446, 474
    diagnosis and, 191
    morbidity and, 83
    mortality and, 85
    prognosis and, 620
Air, stagnant, 698
"Alarm zone," 379, 417
Albumin in sputum, 212
Albuminuria, 287
Alcohol, 854
Allergy, 128, 145
Alopecia, 271
Altitude, artificial pneumothorax and, 810
  frequency of tuberculosis and, 76
  in pthhisiotherapy, 715
Amenorrhea, 192, 576
Amyloid changes, 185
    of intestines, 564
    of kidneys, 287
Amphorophony, 353
Anaphylaxis, 37
Anasarca, 288
Anatomy, morbid, 160
Anemia, 281
Anergy, 128
Anesthesia in artificial pneumothorax, 833
  in phthisical patients, 652
Aneurysms of Rasmussen, 176, 235
Annular shadow, 365
Anorexia, 258
  in advanced phthisis, 260
  causes of, 259
  diet in, 736
  in incipient phthisis, 376
  treatment of, 790
Antagonistic diseases, 584
    asthma, 589
    atherosclerosis, 604
    cancer, 514
    cardiac, 592
    cholelithiasis, 604
    coryza, 588
    gout, 603
    hyperthyroidism, 608
    lymphatism, 588
    nephritis, 604
    nephrolithiasis, 604
    obesity, 606
    scrofula, 584

Antagonistic diseases, syphilis, 598
Anthracosis, 52
in animals, 56
in roentgenogram, 356
Antiformin, 206
Antipyretics, 778
Apex, percussion of, 326
Krönig's method, 325
predisposition of, 117
Apical catarrh, 538
pleurisy, 486
Appendicitis, 612
and pleurisy, 485
Appetite, 258. *See* Anorexia.
Apyretic tuberculosis, 228
Arneth's blood picture, 283
Arrhythmia, 280
Arsenic, 754
Articular tuberculosis, 586
Ascites, 567
Asthenic constitution, 123
Asthma and tuberculosis, 589
Atavistic tendencies, 123
Atherosclerosis, 604
Athletes, tuberculosis in, 640
Atrepsia in infants, 451
Auscultation, 336
in abortive tuberculosis, 417
in advanced phthisis, 400
for adventitious sounds, 345
in aged patients, 476
in artificial pneumothorax, 814, 834
in cancer of the lung, 548
in children, 465
in incipient phthisis, 379
over cavities, 404
in pleural effusions, 494
in pneumothorax, 527
single phase, 337
sources of error in, 343
technic of, 336
of voice sounds, 352
of whispered voice, 352
Autoinoculation, 693
Autonomic nervous system, 290
Autopsies, reliability of statistics of, 68
tuberculous lesions found at, 62
Autoserotherapy, 857
Autoserum test, 393
Autourine test, 393
Avian bacilli, 28

B

Bacilli, tubercle, 17
in abortive tuberculosis, 415, 416
acid-fast, 18, 29
in acute forms of tuberculosis, 433
atypical, 42
avian, 28
in human beings, 29
avirulent, 68
in abortive tuberculosis, 414
in latent lesions, 86
Bacilli, tubercle, in birds, 28, 32
bovine, 27, 32, 58
in children, 33, 663
in human beings, 32, 58
immunity to, 154
mutation of, 41
in phthisis, 38, 158
prophylaxis, 663
virulence in humans, 34, 127
in calcified glands, 144
in circulating blood, 284
in cerebrospinal fluid, 443
channels of entry, 43
classification of, 20
in cold-blooded animals, 29
cultivation of, 20
diagnostic value of, 382
in domestic animals, 32
in dust, 46
in early phthisis, 382
in embryo, 109
examination for, 205
antiformin method, 208
fatty substance in, 18
in fibroid phthisis, 421
in giant cells, 162
in healed lesions, 39, 158
in healthy persons, 51
human, 26
in human milk, 663
infective dose of, 140
in reinfection, 146
ingestion of, 54
inhalation of, 40
inoculation of, 21, 44, 209
in intestines, 54
latency of, 143
media for, 20
microscopic examination for, 204
in milk, 57
morphology of, 17
multiplication in human body, 141
mutation of, 41
non-pathogenic, 29
differentiation of, 31
in non-tuberculous patients, 383, 414
number in sputum, 61, 208, 624
occurrence in nature, 32, 40
in ovum, 109
as parasites, 40
in placenta, 106
in pleural effusions, 509
poisons from, 35
powers of resistance, 21
prognosis and, 208, 624
pseudotubercle, 29
grass, 30
leprosy, 30
smegma, 29
trumpet, 30

Bacilli, tubercle, in sausages, 22
in semen, 106
in serous membranes, 35
sources of, 43
spread within the body, 183
in sputum, 205
prognostic value of, 208, 624
staining of, 18, 205
in stomach, 54
in tonsils, 125
toxins of, 36
transitional, 42
transmutation of, 42
in traumatic tuberculosis, 642
types of, 17, 25, 40
ubiquity of, 61
virulence, 24
in abortive tuberculosis, 414
in acute tuberculosis, 430
in glands, 144
vitality of, 23
zoögenetic, 34
Bacteria, pyogenic, 601
Bacteremia, 284, 601
after tuberculin treatment, 391, 770
in miliary tuberculosis, 437
non-tuberculous, 602
in skin infections, 44
Ballooning, 814, 815
Basal lesions, 540
Baths, 681
Bell sound, 529
Blood, 281
bacilli in, 284
picture, 282
Arneth's, 283
in artificial pneumothorax, 812
changes in mountains, 716
in miliary tuberculosis, 444
in pleural effusions, 505
in stools, 564
Blood-pressure, 281
prognosis and, 624
Bradycardia, 280
Breath sounds, absence of, 339
in advanced phthisis, 401
amphoric, 407
in pneumothorax, 527
bronchial, 342
bronchovesicular, 343
cavernous, 407
in children, 465
cog-wheel, 341
feeble, 338
granular, 340
interrupted, 341
metamorphosed, 408
normal, 338
rough, 340
Bronchiectasis, 542
in fibroid phthisis, 427
in tuberculous lungs, 174
Bronchitis, 589
hemorrhagic, 551
Bronchophony, 352
in children, 467
in pleural effusions, 494
Bronchopneumonia, tuberculous, 433
complications of, 436
diagnosis of, 436
etiology of, 433
hemoptysis and, 255
in infants, 451
physical signs of, 435
prognosis of, 436
symptoms of, 434
tuberculosis and, 591
Brownlee's three types of phthisis, 98
Butter in diet, 742

C

Cachexia, 263
in infants, 451
Calcification, 165
Calcium in diarrhea, 793
in hemoptysis, 786
Cancer of lung, 547
tuberculosis and, 614
Carbohydrates as foods, 743
Carcinomatosis, miliary, 446
Cardiac diseases, 127, 552
weakness, treatment of, 785
Cardiovascular symptoms, 277
"Carriers," 208, 728
Caseation, 164
Caseous pneumonia, 430
Catarrh, apical, 538
Causes, constitutional, 101
Cavities, pulmonary, 173
adventitious sounds over, 408
bacilli in, 600
basal, 409, 540
bleeding from, 176, 239
breath sounds over, 407
bronchiectatic, 174, 427, 542
closed, 176
cough from, 199
diagnosis of, 403
differentiation from pneumothorax, 531
in fibroid phthisis, 421
healing of, 180
mixed infection in, 600
pathology of, 173
phantom, 409
postural treatment of, 771
prognostic significance of, 626
roentgenology of, 365
sputum from, 202
treatment of, 771
tympany over, 404
whispered voice over, 353
Cerebrospinal fluid, 443
Chairs, reclining, 701

Cheese as a food, 741
tubercle bacilli in, 21
Chemotherapy, 747
Chest in advanced cases, 311
in aged patients, 476
appearance in roentgenogram, 354, 357
asthenic, 305
asymmetry of, 308
deformities of, 303
enlarged veins on, 301
flat, 303
life insurance and, 649
muscular changes in, 309
normal, 301
paralytic, 303
phthisical, 302
acquired, 305
in children, 563
congenital, 305
pterygoid, 303
retractions of, 308
Children, pulmonary tuberculosis in, 447
bovine infection in, 33, 663
characteristics of, 447
exposure to infection of, 192, 660
extent of infection among, 70
fat consumption in, 266
meningitis in, 441
prognosis in, 471
prophylaxis in, 660
roentgenography in, 468
scrofula in, 454
symptoms of, 453
reflex, 468
treatment of, 850
tuberculin test in, 387, 470
Chloasma phthisicorum, 268
Chlorosis, pretuberculous, 375
Cholelithiasis, 604
Cholesterin in pleural effusions, 520
Choroid, tubercles in, 445
Cirrhosis of liver, 613
City life and tuberculosis, 77, 97
Civilization and tuberculosis, 61, 76
Classification of phthisis, 368
author's, 371
official, 368
shortcomings of, 369
Climate, infection and, 73, 76
Climates, desert, 721
mountain, 715
contraindications, 718
indications, 717
sea, 719
Climatic treatment, 710
cost of, 711
economic aspects of, 710
effects of, 712
vs. open-air treatment, 697
where obtained, 714
Clothing, 681
Clubbed fingers, 271
Clubbed fingers in fibroid phthisis, 422
Coal miners, immunity of, 132
Cod liver oil, 758
administration of, 750
contraindications, 759
indications, 759
Cog-wheel breath sounds, 341
"Colds," 125
in etiology, 125
in history of the disease, 192
pleurisy and, 482
Collapse during hemorrhage, 239
induration, 537
in pneumothorax, 523, 816
treatment of, 788, 817
Coma, 442
Complement-fixation test, 392
prognostic value of, 629
Complexion, 123, 267, 299
Complications of phthisis, 558
abscess of chest wall, 574
appendicitis, 612
cardiac, 568
empyema, 506
gangrene of lung, 563
influenza, 595
intestinal tuberculosis, 564
laryngeal tuberculosis, 558
meningitis, 441
myocarditis, 568
nephritis, 572
pericarditis, 568
peritonitis, 565
phlebitis, 569
pleural effusions, 505
pleurisy, dry, 500
pneumothorax, 521
prognosis and, 625
purpura, 573
pyelitis, 572
terminal edema, 572
thrombosis, 569
influence on prognosis, 413
tongue, ulcerations of, 573
treatment of, 856
ulcerations of mucous membranes, 573
urogenital tract, 572
Condiments in diet, 744
Congenital infection, 108
Conjugal phthisis, 149
Constipation, 262
treatment of, 791
Constitutional diseases, 602
inferiority, 122
Contact infection, 44
Convulsions, 443
Corsets, 681
Coryza, rare in tuberculosis, 588
Cough, 195
in abortive tuberculosis, 416
absence of, 195
in acute pneumonic phthisis, 431
in advanced phthisis, 199, 398
in aged patients, 475

Cough in bronchial adenopathy, 457, 460
in bronchiectasis, 199
in children, 460
diagnostic significance of, 200
in early stages, 195
emetic, 197
treatment of, 774
in fibroid phthisis, 425
frequency of, 195
hysterical, 196, 200
in incipient phthisis, 475
in infants, 457
infection through, 48
paroxysmal, 196
prognostic significance of, 200
psychic influences on, 200
psychotherapy of, 772
treatment of, 771
medicinal, 773
in tuberculous bronchopneumonia, 435
"Cough phenomenon," 359
Cracked-pot resonance, 406
Creosote, 749
administration, 751
contraindications, 751
derivatives from, 752
indications, 751
Crepitation, 346
Cuspidors, 671

D

Dactylitis, tuberculous, 455
Death, modes of, 412
from pulmonary hemorrhage, 253
in laryngeal tuberculosis, 562
in pleurisy, 508
premonitory signs of, 366
rates from tuberculosis, 75, 76, 79, 82
Degeneration, stigmata of, 122
Delirium, 295, 441, 443
Demineralization, 112
Dermographism, 267, 290
Desert climate, 721
D'Espine's sign, 466
Dextrocardia, 401
Diabetes, 604
artificial pneumothorax and, 833
rarity in tuberculous, 606
symptoms of, 605
Diagnosis by animal inoculation, 209
dangers of hasty, 187
differential, 534
from abscess of the lung, 546
from actinomycosis, 549
from acute endocarditis, 554
pneumonic phthisis, 433
from apical catarrh, 538
from bronchiectasis, 542
from bronchopulmonary spirochetosis, 551
Diagnosis, differential, from cancer of the lung, 547
from cardiac disease, 552
from chronic bronchitis, 541
pneumonic processes, 539
from collapse induration, 537
from gangrene of the lung, 546
from hemorrhagic bronchitis, 551
from hyperthyroidism, 556
from influenza, 595
from mitral stenosis, 552
from neurocirculatory asthenia, 557
from nonspecific pulmonary infections, 539
from pericarditis, 554
from pleural vomicæ, 544
from pulmonary infarction, 554
streptothrichosis, 550
from rhinopharyngeal diseases, 535
from syphilis of the lung, 554
from tonsillitis, 536
elementary principles of, 190
history in, 191
natural method of, 191
Diaphragm in pneumothorax, 530
roentgenography of, 359
Diaphragmatic pleurisy, 483
Diarrhea, 262, 564
in aged patients, 476
due to diet, 741
emaciation and, 265
treatment of, 792
Diathesis, 122, 141
arthritic, 603
Diazo-reaction, 629
Diet, 733
for febrile patients, 778
in hemoptysis, 787
in sanatoriums, 730
vegetarian, 738
weight and, 734
Dietaries, 745
Dietetic treatment, 733
economic aspects of, 733
individualization in, 733
need for special, 736
variety in, 736
Disability, occupation and, 637
resulting from tuberculosis, 634
Disease *vs.* infection, 61, 101, 187, 454
Diseases, preëxisting, 192
Domestic servants, selection of, 661
Droplet infection, 48
Duration of life, 411
Dust, 131
bacilli in, 46
coal, 132
effects on lungs, 46, 51, 133
in etiology of fibroid phthisis, 420
harmlessness of, 132
Dusty trades, 131
Dyspepsia, 257

Dyspepsia in advanced phthisis, 261
frequency of, 257
Dysphagia, 559
in artificial pneumothorax, 820
treatment of, 862
Dysphonia, 559
Dyspnea, 278
in artificial pneumothorax, 813
in cancer of the lung, 547
cardiac, 593
as a danger signal, 689
in fibroid phthisis, 427
high altitude and, 718
in infants, 452
in miliary tuberculosis, 440
in pneumothorax, 523
treatment of, 788

E

Economic conditions in etiology, 81, 89, 92
in prognosis, 630
treatment and, 679
Edema, angioneurotic, 267
cachectic, 571
in fibroid phthisis, 427
of legs, 570
terminal, 288, 413, 572
Effusion. *See* Pleural effusion.
Eggs, diarrhea from, 262, 742
in diet, 741
Egotism, 295
Elastic fibers in sputum, 210
Ellis's line, 492
Emaciation, 263
in acute phthisis, 432, 435
in advanced phthisis, 399
in aged patients, 475
in arrested disease, 632
in children, 458
effects of, 263
extent of, 263, 432
in fibroid phthisis, 422
in glandular tuberculosis, 458
in incipient phthisis, 376
in infants, 451
prognostic significance of, 234
seasonal influences, 265
Embolism, gas, in artificial pneumothorax, 817
pulmonary, 552
Embryo, tubercle bacilli in, 109
Emetin in hemoptysis, 784
Emphysema in artificial pneumothorax, 819
pathology of, 180
pneumothorax and, 522
pulmonary tuberculosis and, 596
subcutaneous, 820
subfascial, 820
Empyema, 506
in cancer of lung, 506, 549
prognosis in, 519
Empyema, treatment of, 857
Endemic diseases in etiology, 127
Endocarditis, 185
Endocrine glands, dysfunctions of, 113, 607
Endotoxins, 36, 762
Epidemiology, 61
Epididymitis, 587
Epithelioid cells, 162
origin of, 163
Erythema induratum, 45
nodosum, 585
Erythrocytes, 281
Eugenics, tuberculosis and, 111, 676
Eunuchism, 115, 587
Euphoria, 295
in advanced phthisis, 399
Euthanasia, 295
Exanthemata and tuberculosis, 594
Exercise, 692
effects on temperature, 218
Exotoxins, 762
Expectation of life, 411
Expectorants, 775
Expectoration, 201
in children, 201
treatment of, 775
Exposure to infection, 634, 648
of infants, 450, 663
life insurance and, 634, 648
Extrapleural pneumolysis, 841
Extrapulmonary tuberculous lesions, 584

F

Facies, 299
Familial tuberculosis, 450
Family history, insurance and, 644
Farming, 684
Fat consumption, 266, 607
in diet, 742
intolerance of, 258
Fecundation, 579
Fertility, 298, 579
Fetus, infection of, 108
Fever, 213
in abortive tuberculosis, 416
absence of, 228
in acute pneumonic phthisis, 432
in advanced phthisis, 399
after artificial pneumothorax, 811, 823
after hemoptysis, 255
in aged patients, 475
anorexia and, 218, 258
antipyretics in, 778
in children, 537
chronic, in non-tuberculous diseases, 223
continuous, 225
cyclic, 225
diagnostic significance of, 229
in differential diagnosis, 536
due to complications, 229

Fever due to medication, 229
effects of rest on, 690
evaluation of, 221
exercise and, 689, 692
in fibroid phthisis, 422, 425
in glandular tuberculosis, 458
hectic, 226
hydrotherapy in, 778
hysterical, 223
in incipient phthisis, 217, 222, 376
influence of hemoptysis on, 254
intermittent, 226
irregular, 227
medication for, 778
menstrual, 221
in miliary tuberculosis, 439, 444
mixed infection in, 213
mountain climate for, 717
open-air treatment for, 704
in pleurisy, 489, 501
premenstrual, 221
prognostic significance of, 229, 622
provoked, 218
psychic state and, 220
pulse in, 278
reaction to, 228
rest and, 689, 692
reverse type of, 223
as a symptom of activity, 213
symptoms of, 218
in tracheobronchial adenopathy, 459
treatment of, 776
in tuberculin reaction, 390
in tuberculous bronchopneumonia, 434
types of, 224
undulating, 225
Fibroid phthisis, 419
course of, 423
diagnosis of, 424
emphysematous form, 422
etiology, 420
forms of, 421
pathology of, 421
physical signs in, 423
pleural form, 426
prognosis in, 428
simple fibrosis, 424
syphilis and, 420, 599
treatment of, 850
Fingers, clubbed, 271
First born, handicap of, 110
Fish in diet, 739, 743
Fistula-in-ano, 613
Fluoroscopy, 357
of children, 469
Folliclis, 270
Foods, carbohydrate, 743
condiments in, 744
protein, 738
Forced feeding, 734
dangers of, 743, 744
precautions in, 736
Friction sounds, 350
differentiation from rales, 487
Friedreich's phenomenon, 405
Fumes, inhalation of noxious, 136

G

Gabbet's stain, 19
Galloping consumption, 430
Games, indoor, 695
outdoor, 695
Gangrene of the lung, 563
Gastro-intestinal symptoms, 257
in advanced phthisis, 260
treatment of, 791
tuberculosis and, 612
Genito-urinary organs, 184
pulmonary tuberculosis and, 587
tuberculosis of, 587
Genius, tuberculosis and, 297
Geographical distribution, 76
Gerhardt's phenomenon, 406
Germinative transmission, 107
Giant cells, 160
Glands, cervical, in apical pleurisy, 488
enlarged, 301
infection of, 57, 64
mesenteric, 184
pathology of, 168, 184
pulmonary tuberculosis and, 586
tracheobronchial, 168
in roentgenogram, 356
tuberculous. *See* Adenopathy.
virulent bacilli in, 144
Glycosuria, 606
Gonads, 610
in etiology, 115
Gout, fibroid phthisis and, 420
tuberculosis and, 603
Graduated labor, 693
Graves's disease, 608
Grocco's triangle, 493
Growth, 575
Guinea-pigs, spontaneous tuberculosis in, 210

H

Habitus phthisicus, 299
in children, 463
Hair, 271
color of, 123
Handkerchiefs, 673
Hardening, 705
of children, 851
Headache, 442
Head's zones, 292
Heart, diseases of, 592
displacement of, 401, 410, 426
effects on tuberculosis, 593
hypertrophy of, 592
irritability of, 278
palpitation of, 277
pathology of, 185

Hectic fever, 226
Hematemesis, 251
Hematogenous infection, 53
Hemophobia, 248
Hemoptysis, 234
in abortive tuberculosis, 416
in abscess of lung, 546
in acute pneumonic phthisis, 432
respiratory diseases, 245
in advanced phthisis, 238, 400
in aneurysm of the aorta, 246
in arrested disease, 631
arthritic, 244
in artificial pneumothorax, 812, 829
blood-pressure and, 281
in bronchiectasis, 246, 250, 258, 543
in bronchitis, 243
bronchopneumonia after, 435
causes of, 241
convalescence from, 787
deaths due to, 253
diagnostic significance of, 243
diet in, 787
disability from, 636
during lactation, 248
menopause, 855
tuberculin treatment, 720
effects of, 236
epidemics of, 242
excitement and, 780
exertion and, 241, 636, 780
fatality of, 253
in fibroid phthisis, 424, 427
false, 244
frequency of, 234
from esophagus, 244, 247
in heart disease, 245, 552
hematemesis and, 251
hereditary, 249
high altitude and, 719
hysterical, 248
influence of, on course of disease, 254
in influenza, 245
life insurance and, 647
localization of source of, 250
menstrual, 247, 577
of nervous origin, 248
in non-tuberculous diseases, 252
at onset of phthisis, 237, 374, 377
overexertion and, 241, 636, 780
pathology of, 177, 235
in pleurisy, 245, 489
in pregnant women, 248
premonitory symptoms of, 238
prognostic significance of, 252, 624
prophylaxis of, 780
in pulmonary emphysema, 245
infarction, 246
spirochetosis, 551
in rhinopharyngeal conditions, 244
seasonal influences on, 242
sexual differences and, 241
spurious, 244
Hemoptysis, stature and, 241
streaky, 243
symptoms of, 239
in syphilis of lung, 246
traumatic, 640
treatment of, 780
by artificial pneumothorax, 783, 829
posture during the, 781
tying the extremities in, 783
venesection in, 786
with adrenalin, 784
with atropin, 785
with blood serum, 786
with calcium salts, 786
with camphor, 786
with digitalis, 785
with emetin, 784
with ergot, 785
with gelatin, 785
with morphine, 782
with nitrites, 784
with salt, 782
of unknown origin, 249
Hemorrhages, intestinal, 564
Hemorrhagic phthisis, 240
Heredity, 103
biological aspects of, 105
clinical aspects of, 111
definition of, 105
germinative, 105
statistics of, 103
Hermann stain, 205
Herpes zoster, 268
Hilus shadow, 356, 358, 362
in children, 569
History of exposure, 192
in infants, 449
of the patient, 191
of the present illness, 192
prognostic significance of, 621
Hoarseness, 201
in early phthisis, 377
in laryngeal tuberculosis, 560
in mitral stenosis, 553
House infection, 46
Hydropneumothorax, 527
roentgenography of, 366, 531
treatment of, 859
Hydrotherapy, 778
Hygiene, personal, 680
Hyperesthesia, 292
in pleurisy, 483
Hypersensitiveness to foreign proteins, 37, 390
phenomena of, 37
Hyperthyroidism, 608
differential diagnosis of, 556
effects on tuberculous disease, 607
in etiology, 144
Hypoasphyxial syndrome, 277
Hypotension, arterial, 281
adrenals in, 115, 609
in early phthisis, 377
Hypothyroidism, 609

I

Ichthyol, 754
Idiocy, 294, 616
Immunity, 138
  acquired by infection, 145
  of adults, 146, 158
  clinical evidence of, 151
  of consorts, 149, 674
  endogenous, 146
  exogenous, 145
  experimental proof of, 145
  failure of, 155
  "father," 150
  of hospital staffs, 147
  "mother," 150
  of mucous membranes, 151
  of nurses, 147
  phenomena of, 138
  phthisis a manifestation of, 153
  of physicians, 147
  of scrofulous children, 151, 454, 586
  through bovine infection, 154
Immunization, artificial, 666
  passive, 770
  with milk, 59
Incipient phthisis. *See* Phthisis.
Incubation, period of, 451
Indians, American, tuberculosis among, 73
India-rubber ball sound, 408
Infancy, tuberculous during, 449
    diagnosis of, 452
    history of exposure in, 450
    prognosis in, 452
    prophylaxis in, 660
    symptoms of, 451
Infantilism, 576, 611
Infants of tuberculous mothers, 583
    extent of infection among, 70
    newborn, freedom from infection of, 63, 83
Infarction, pulmonary, 554
Infection, tuberculous, 40
  of adults, 146
  age influence on, 62
  barriers against, 50
  "benevolent," 668
  channels of entry, 43, 52
  of children, 58, 62, 70, 147, 660
  congenital, 107
  of consorts, 149
  by contact, 44
  dangers of, 637
  droplet, 48
  experimental, 40
    in human beings, 152
    vs. epidemiological, 59
    vs. spontaneous, 130
  exposure to, 192, 637
    of infants, 663
    life insurance and, 648
  extent of, 70
  familial, 664
Infection, tuberculous, focal, 68
  frequency of, 62
  of glands, 33, 57
  hematogenous, 53
  of hospital staffs, 147
  housing conditions and, 47, 82
  hygienic conditions and, 92
  immunity conferred by, 59, 145
  inadequacy of, 101
  of infants, 49, 63, 70, 142, 664
  by ingestion, 54
    dose in, 141
  by inhalation, 46, 141
  by inoculation, 44, 140
  intensity of, 140
  intraovular, 106
  intrauterine, 106, 108
  latency of, 57, 63, 65, 143, 166
    lymphoid, 144
  lymphogenous, 53
  marital, 149, 674
  metastatic, 144
  mixed, 600
  of nurses, 147
  of physicians, 148
  placental, 107
  prevention of, 660
  of primitive peoples, 73
  problems of, 40
  results of, 102
  in sanatoriums, 148
  sanitary conditions and, 92
  secondary, 601
  spermatogenic, 106
  through cough, 48
    dust, 46, 51
    milk, 663
    mucous membranes, 51
    sputum, 46
    sweat, 233
    the air, 48, 50
    the nose, 50
    the skin, 44
    the sputum, 46
    tonsils, 55
  vs. disease, 61, 101, 187, 454
  vs. spontaneous tuberculosis, 138
  with bovine bacilli, 58, 663
    immunity through, 154
Influenza, 539
  in etiology, 128
  prognosis and, 625
  sequels of, 542
  tuberculosis and, 595
Injury. *See* Traumatic tuberculosis, 638
Insanity and tuberculosis, 616
Insomnia, 297
  treatment of, 789
Inspection, 299
  in children, 463
  in incipient phthisis, 377
  technic of, 305
Institutional treatment, 723. *See* Sanatoriums.

Intellect of consumptives, 296
Internal secretions, 113, 607
Intestines, tuberculosis of, 564
diagnosis of, 564
emaciation and, 265
pathology of, 181
symptoms of, 181
treatment of, 792
Ischiorectal abscess, 182, 613

J

Joints, bovine bacilli in, 33
pulmonary tuberculosis and, 586
tuberculosis of, 84, 455, 586

K

Kidneys, 285
amyloid, 185
symptoms of, 287
tuberculosis of, 572, 587
Krönig's resonant areas, 325
in incipient phthisis, 378

L

Labor, effects on the disease, 582
Lagging, 308
Languor, 376
Larynx, artificial pneumothorax and, 832
tuberculosis of, 558
diagnosis of, 560
frequency of, 558
pathology of, 181
prognosis in, 562
symptoms of, 559
treatment of, 860
Latency of tuberculosis, 51, 57, 63, 65, 68, 143
Latent tuberculosis, avirulent bacilli in, 69
virulent bacilli in, 68
Lesions, tuberculous, among healthy, 62
initial, 52
repair of, 178
Leukocytes, 282
Life insurance aspects, 634
medical examination for, 643
Lime starvation, 112
Lips, tuberculous ulceration of, 573
Liver, pathology of, 186
Locus minoris resistentiæ, 111
Lumbar puncture, 443
Lung, abscess of, 546
apex, predisposition of, 117
bacteriology of, 50
blocks, 83
cancer of, 447
cavities in, 173, 403, 627
circulation in the, 116
as a culture medium, 116
Lung, extension of lesion in, 170
first lesion in, 170
gangrene of, 546
gross appearance of, 167
mixed infection and, 174
perforation of, 827
reparative processes in, 178
in roentgenogram, 358
rupture of, 178, 521, 827
size of the, 116
tubercles in, 167
Lupus, 584
Lymphatism, 588
abortive tuberculosis and, 414
pulmonary phthisis and, 588
scrofula and, 455
Lymphoid latency, 144

M

Malaria complicating phthisis, 230
Manometer, 800
Manometric hints, 805
Marital phthisis, 149, 674
Marriage of tuberculous, 579, 674
Marriages, consanguineous, 123
Measles in etiology, 127
tuberculosis and, 598
Meat in diet, 738
eating, tuberculosis and, 604
raw, 738
Mediastinum, displacement of, 410, 426
in pleural effusions, 499
in pneumothorax, 525, 530, 814
weak, 814
Medication, harmless, 747
Medicinal treatment, 746
importance of, 746
Medico-legal aspects, 634
Meningitis, tuberculous, 441
Menopause, obesity during, 266
tuberculosis during, 855
Menstruation, disturbances of, 577, 610
fever and, 221
hemoptysis during, 247, 577
vicarious, 248, 577
Mental traits, 296
Mercury, 757
Metabolism, calcium, 113
disturbances in, 112
Metallic tinkle, 409
in pneumothorax, 528
Milk, anorexia and, 790
as a cause of diarrhea, 262
dangers of, 24
in diet, 740
human, tubercle bacilli in, 663
infections, 154
injections of, 57
immunization with, 59, 154
pasteurization of, 663
tubercle bacilli in, 21, 30
Miners, rarity of tuberculosis among, 133
Mitral stenosis, 127, 594

Mixed infection, 600
in artificial pneumothorax, 822
excavation and, 174
in pleurisy, 496
Morbidity, 61, 83
in children, 454
vs. infection, 61, 101, 187, 454
Morphine in hemoptysis, 782
Mortality, age effects on, 83, 85, 448, 474
periods of, 448
in American cities, 76
birth rates and, 111
in cities, 77, 79
in the country, 77
decline of, 91
causes of, 91, 96
during childhood, 583
in dusty trades, 132
economic influences, 81, 89, 97, 136
effects of special campaign on, 93
general mortality and, 94
housing and, 82
influence of sex on, 78, 86, 89
natural selection and, 96, 105
occupation and, 89
poverty and, 81, 97
race influences on, 97
sexual differences in, 86
statistics of, 74, 90
urbanization and, 97
in various cities, 79
countries, 79
wages and, 81, 89, 98, 136
Mountain climates, 715
Much's granules, 19
staining of, 208
Murmurs, cardiac, 593
Muscles, hyperexcitability of, 313
pathology of, 185
spasm of, 309
Myocarditis, 568
Myoidema, 312
Myxedema, 609

N

Nails, 272
Natural selection in tuberculosis, 96, 105
Negroes, African, 74
American, 24
Nephritis, 287, 604
Nephrolithiasis, 604
Nervous symptoms, 289
system, autonomic, 290
effects of tuberculosis on, 185
Neurasthenia, 289
Neurocirculatory asthenia, 556
Nightsweats, 231
in aged patients, 476
causes of, 231
in children, 459
in incipient tuberculosis, 377
symptomatology of, 232
treatment of, 779
Nocardia, 550
Nose, tubercle bacilli in, 51
Nursing of infants, 661

O

Obesity, 266, 606
Occupation, 129
diagnosis and, 191
disability and, 637
dusty, 131
indoor *vs.* outdoor, 685
suitable for tuberculous, 683
Ochrodermia, 282
Onset of phthisis, 192, 374
acute, 431
prognosis and, 622
with hemoptysis, 237
with pleurisy, 374, 482
Open air schools, 852
treatment, 696
of children, 851
contraindications, 707
of febrile patients, 704
results obtained from, 706
technic of, 699
vs. climatic, 697
where obtainable, 696
Operations, surgical, 625, 642, 833
Ophthalmoreaction, 389
Opiates for cough, 774
Opsonic index, 283, 395
Optimism, 296
Osseous tuberculosis, 586
Osteoarthropathy, pulmonary, 274
Overfeeding, 734
precautions necessary while, 737
symptoms of, 737
Ovum, tubercle bacilli in, 109
Ozone, 715

P

Pains, 291
in artificial pneumothorax, 818
in chest, 291
treatment of, 789
in pleurisy, 483
Palpation, 299
"light touch," 307
technic of, 305
for vocal fremitus, 312
Palpitation, cardiac, 277
causes of, 278
Parrot's law, 168
Pasteurization of milk, 663
Pathology, 160
Pectoriloquy, 409
Percussion, 314
in abortive tuberculosis, 417
in advanced phthisis, 400
in aged patients, 476
aims of, 314

Percussion, apical, 325
auscultation and, 314
in bronchial adenopathy, 465
in children, 465
comparative, 320
diagnostic value of, 335
hooked-finger, 320
in incipient phthisis, 323, 378
over a cavity, 404
in pleural effusions, 490
pleximeter finger in, 318
in pneumothorax, 527
respiratory, 324
sources of error in, 329, 334
technic of, 316
tidal, 332, 490
in pleurisy, 491
topographical, 325
Percutaneous tuberculin test, 388
Pericarditis, 568
Perichondritis, 559
Peritoneum, pathology of, 182
Peritonitis, tuberculous, 565
acute, 566
adhesive, 567
chronic, 566
pathology of, 182
symptoms of, 566
Personal hygiene, 680
Phlebitis, 569
Phototherapy, 708
Phrenikotomie, 843
Phthisiogenesis, 101, 138
Phthisiophobia, 669
Phthisis acquired during childhood, 142
acute forms of, 429
pneumonic, 430
course of, 432
diagnosis of, 433
physical signs of, 432
symptoms of, 431
advanced, 396
afebrile, 399
auscultation in, 400
duration of, 410
euphoria in, 399
oscillating course of, 397
physical signs of, 400
symptoms of, 398
treatment of, 845
in aged, 474
course of, 477
diagnosis of, 477
etiology of, 474
frequency of, 474
physical signs of, 476
symptoms of, 476
treatment of, 854
classification of, 272, 368
clinical forms of, 367
curability of, 618
a distinctly human disease, 139
factors predisposing to, 101
fibroid, 419. *See* Fibroid phthisis.
hemorrhagic, 240
Phthisis, hyperthyroidism and, 556
incipient, 374
course of, 396
curability of, 618
diagnosis of, 381
complement-fixation test in, 392
elements of, 381
roentgenography in, 393
sources of error in, 381
treatment of, 844
tuberculin test in, 384
latency of, 143
a manifestation of immunity, 153
marital, 149, 674
natural resistance against, 414
onset of, 474
oscillating course of, 397
pathology of, 167
physical signs of, 377
polymorphism of, 367
rarity in children of, 477, 665
remissions in, 397
scrofula and, 455
stages of, 368
stigmata of, 299
traumatic, 639
treatment of, 844
Pityriasis tabescentium, 269
versicolor, 269
Placenta, tubercle bacilli in, 108
Placental transmission, 108
Pleura, accommodative powers of, 525
anatomy of, 478
infection of, 578
pathology of, 181, 479
in roentgenogram, 366
rupture of cavity into, 178, 523
tuberculosis of, 478, 481
Pleural adhesions, 502
artificial pneumothorax and, 834
diagnosis of, 502, 834
pathology of, 480
roentgenography of, 583, 834
thoracoplasty in, 842
thoracoscopy for, 835
effusions, 489
in acute phthisis, 498
in artificial pneumothorax, 821
in cancer of the lung, 548
in chronic phthisis, 501, 505
symptoms of, 501
during the course of phthisis, 505
exploratory puncture in, 495
hemorrhagic, 505
interlobar, 497
physical signs of, 490
in pneumothorax, 527
primary, 489
purulent, 506
prognosis in, 508 519
symptoms of, 506,
in roentgenogram, 366

Pleural effusions, tubercle bacilli in, 510
shock, 816
vomicæ, 544
Pleurisy, 481
in acute phthisis, 499
apical, 486, 488
appendicitis and, 612
in artificial pneumothorax, 821
beneficial, 515
in chronic phthisis, 500
diaphragmatic, 483
dry, 481
exudate in, 495
cytology of, 496
fatal, 508
hemoptysis in, 245
idiopathic, 482
initial, 482
interlobar, 485
life insurance and, 646
non-specific, 514
at onset of phthisis, 374
pains in, 483
primary, 481, 508
prognosis in, 507, 515, 518
recurrent, 486, 488
roentgenography of, 496
statistics of, 512
sudden death in, 509
traumatic, 639
treatment of, 856
varieties of, 481
with effusion, 489
course of, 498
physical signs of, 490
Pleximeter, 318
hooked finger, 320
Pneumokoniosis and lung apex, 117
roentgen findings in, 446
tuberculosis and, 131
Pneumonia, apical, 433
broncho-, tuberculosis and, 591
caseous, diagnosis of, 433
pathology of, 169
prognosis in, 620
symptoms of, 431
lobar and tuberculosis, 591
Pneumoperitoneum, 821
Pneumothorax, 521
artificial, 794
absorption of gas in, 809
in advanced phthisis, 829
age of the patient and, 833
altitude and, 810
apparatus for induction of, 798
bilateral, 836
blood picture in, 812
Brauer's method, 797
cardiac disease and, 833
cases suitable for, 835
complications of, 815, 838
active lesion in untreated side, 828
during operation, 816
emphysema, 819
Pneumothorax, artificial, complications of, emphysema, deep, 820
interstitial, 821
mediastinal, 820
subcutaneous, 819
subfascial, 820
empyema, 823
gas embolism, 817
hemoptysis, 830
pains, 818
perforation of the lung, 827
pleural effusion, 821
bilateral, 824
septic, 823
symptoms of, 822
treatment of, 826
shock, 818
spontaneous pneumothorax, 818
contraindications, 832
diabetes and, 833
diagnostic, 831
duration of treatment, 836
dyspnea in, 813
fever in, 823
Forlanini method, 797
frequency of refills, 810
gas embolism in, 817
used for inflation, 802
for hemoptysis, 783
hemostatic effects of, 812
history of, 794
in incipient cases, 832, 839
indications for, 828
induction of, 797
injection in, selection of point for, 803
intrapleural pressure in, 800
in laryngeal tuberculosis, 832
limitations of, 837
local anesthesia in, 804
manometer in, 800, 805
manometric hints in, 805
Murphy's method, 797, 808
needle for, 802
open incision method, 797
partial, 836
perforation of lung in, 827
physical signs of, 813
pleural adhesions and, 834
shock in, 818
pneumoperitoneum in, 821
pregnancy and, 833
principles underlying treatment, 795
proportion of cases suitable for, 835
pupils in, 291
pyothorax in, 823
treatment of, 826
roentgenography in, 815
selection of cases for, 828
surgical operations and, 833
symptoms of, 810

Pneumothorax, artificial, technic of, 797
of insufflation, 805
of refilling, 808, 810
in urgent cases, 808
thoracocentesis in, 804
ultimate results in, 839
spontaneous, 521
in artificial pneumothorax, 818
diagnosis of, 528
displacement of organs in, 524
double, 526
effusion in, 527
frequency of, in phthisis, 523
latent, 526
localized, 531
mechanism of, 524
mute, 526
partial, 526
pathology of, 524
physical signs of, 526
prognosis in, 533
"providential," 523
recurrent, 522
roentgenography in, 530, 532
symptoms of, 523
traumatic, 639
treatment of, 858
valvular, 524
Poisons of tubercle bacilli, 35
Polyserositis, 509
Polyuria, 286
Post-tussive suction sound, 408
Poverty, prognosis of phthisis and, 81, 630
Predisposition, 101
anatomical factors and, 115
constitutional factors and, 101
environmental factors, 101
hereditary, 103
of the lung, 115
apex, 117
nature of, 155
occupational, 129
stigmata of, 122
Pregnancy, 579, 675
artificial pneumothorax and, 833
prevention of, 675
Preëxisting diseases, 124
acute infectious, 127, 594
asthma, 126
bronchial, 126
cardiac, 137
influenza, 128, 595, 625
measles, 127, 595
pleurisy, 125, 481
pulmonary, 126
syphilis, 599
typhoid fever, 129, 598
whooping cough, 127
Prognosis, 618
in abortive tuberculosis, 414, 619
activity of the disease in, 622
in acute forms of tuberculosis, 620
age in, 620
in arrested disease, 631
Prognosis, bacilli in sputum and, 624
cavities and, 626
in children, 471
complement-fixation test in, 629
complications and, 624
diarrhea and, 625
economic conditions and, 630
elements of, 619
emaciation and, 264, 632
extrapulmonary tuberculous lesions and, 584
fever and, 229, 622
glandular tuberculosis and, 586
hemoptysis and, 252, 624
heredity and, 621
history of patient and, 621
importance of, 618
in infants, 452
intestinal tuberculosis and, 625
labor and, 582
in laryngeal tuberculosis, 56
onset of the disease and, 622
physical signs in, 626
pleurisy and, 625
pregnancy and, 579
puerperium and, 582
pulse rate and, 623
sex and, 621
side affected and, 627
special tests in, 629
surgical operations and, 625
symptomatology and, 622
tuberculous kidneys and, 587
skin lesions and, 584
in various forms of phthisis, 619
Prophylaxis, 660
in adults, 667
by artificial immunization, 666
in children, 664
of disease, 668
duties of the community in, 673
failure of, 95
in infants, 661
of infection, 660
marriage and, 674
of reinfection, 665
Protein foods, 738
Pseudotubercle bacilli, 29
Psychasthenia, 289
Psychic traits, 294
Psychotherapy, 656, 658
of cough, 772
with medication, 747
with tuberculin, 768
Puberty, 576
in etiology, 115
tuberculosis and, 611
Pulmonary osteoarthropathy, 274
Pulse rate, 278
instability of, 278
prognosis and, 623
Puncture, exploratory, 495
Pupil in acute miliary tuberculosis, 443
in apical pleurisy, 481, 486
in artificial pneumothorax, 291

Pupil, dilatation of, 290
Purpura, 573
Pyelitis, 572
Pyopneumothorax, 506, 823

Q

Quartz lamp in therapy, 708

R

Racial mixture and tuberculosis, 123
susceptibility, 73
Rales in abortive tuberculosis, 417
in advanced phthisis, 402
after hemoptysis, 402
atelectatic, 351
consonating, 348
crepitant, 346
in incipient phthisis, 380
latent, 349
moist, 347
localization of, 348
over cavities, 408
pleural effusions, 494
in pneumothorax, 831
provoked, 349
sibilant, 349
sonorous, 349
spurious, 351
Reinfection, 145
autogenous, 156
endogenous, 146, 156
exogenous, 145, 156
in hospital inmates, 147
metastatic, 156
modes of, 147
prevention of, 665
Renal function, 286
Rest cure, 687
contraindications for, 691
exercise and, 688
indications for, 688
principles of, 687
technic of, 691
Rheumatism and tuberculosis, 602
Roentgenography, 354, 361
in acute miliary tuberculosis, 445
in advanced phthisis, 365
of apex, 359, 361
in artificial pneumothorax, 815
cavities in, 365
in children, 468
diaphragm in, 359
of hilus shadow, 356, 358, 362
in incipient phthisis, 383
in interlobar effusions, 504
limitations of, 364
lungs in, 358
normal chest in, 354, 357
of pleura, 496
of pleural adhesions, 503
in pleurisy, 496
Roentgenography in pneumothorax, 530, 532
sources of error in, 363
in tracheobronchial adenopathy, 468
Roentgen rays in therapy, 709

S

Salt in diet, 743
Scarlet fever, 594
Sclerosis, 166
Scrofula, 454, 586
prognosis in, 456
relation to pulmonary tuberculosis 456, 586
Sanatoriums, 723
abortive tuberculosis in, 415
causes of failure of, 729
cost of, 727
diet in, 730, 733
educational value of, 728
inadequacy of, 725
indications for, 731
infection in, 148
lasting results in, 726
life insurance and, 647
non-tuberculous cases in, 415, 584
prophylactic value of, 728
scope of, 723
Sea climates, 719
voyages, 720
Sex glands in etiology, 115
hemoptysis and, 241
mortality according to, 86
in prognosis, 621
Sexual characters, secondary, 577
excesses, 578
functions, 578
sphere, 298
Semen, tubercle bacilli in, 107
Shoulder, pain in, 488
Silicosis, 135
Skin, 266
bronzing of, 114, 268
eruptions, 270
immunity of, 45
infection through, 44
inspection of, 301
lesions, tuberculous, 584
Smallpox, 595
Smegma bacilli, 29
Smith's sign, 467
Smoking, 682
Softening of lesion, 165
Specific treatment, 761
Spermatogenic infection, 106
Spes phthisica, 296
Spina ventosa, 455
Spinal muscles, sign of, 490
Spirochetosis, bronchopulmonary, 551
in gangrene of the lung, 563
Spleen, 185
Sputum, 202
in abortive tuberculosis, 416

Sputum in abscess of the lung, 546
in advanced phthisis, 202
albumin in, 212
animal inoculation of, 209
bacilli in, 204
chemical examination of, 212
collection of specimen, 204
cytology of, 212
disposal of, 671
elastic fibers in, 210
examination of, 204
fetid, 546, 563
in fibroid phthisis, 423
flasks, 672
in gangrene of the lung, 546, 563
inhalation of, 46
microscopic examination of, 204
spirochetes in, 551
streaky, 243
in arrested disease, 632
Stature, 575
hemoptysis and, 241
Status bacillaris, 109
thymico-lymphaticus, 588
Stigmata of phthisis, 299
Stomach, dilatation of, 260
tubercle bacilli in, 54
Stomatitis, aphthous, 413
Stone in gall-bladder, 604
in kidney, 604
Street-sweepers, rarity of tuberculosis among, 133
Streptothrichosis, pulmonary, 550
Succussion sound, 528
Suggestion, amenability to, 295
in treatment, 657
with tuberculin administration, 657, 769
Sunlight, artificial, 708
Superalimentation, 734
dangers of, 743, 744
precautions in, 737
Surgical operations, traumatic tuberculosis and, 642
in tuberculous, 625
in artificial pneumothorax and. 833
Sweats, 231
Symptomatic treatment, 771
Symptomatology, 187
importance of, 194, 370
Symptoms, constitutional, 190, 370, 536
Syphilis and fibroid phthisis, 420
of the lung, 554
and tuberculosis, 598

T

Tachycardia, 278
in abortive tuberculosis, 416
causes of, 280
high altitude and, 718
in incipient phthisis, 377
paroxysmal, 279
Tachycardia, permanent, 279
prognosis and, 623
treatment of, 788
Tattooing, infection through, 44
Temperature, 213. *See* Fever.
frequency of taking, 215
instability of, 220
normal, 216
oral *vs.* rectal, 214
subnormal, 227, 231
technic of taking, 214
types of, in phthisis, 224
Tents, 699
Thermometers, 213
Thoracoplasty, 841, 842
Thoracoscopy, 835
Thorax. *See* Chest.
asymmetry of, 308
deformities of, 303
normal, 301
phthisical, 302
stenosis of upper aperture of, 118
Thrombosis, 569
of femoral vein, 570
diagnosis of, 571
of jugular vein, 571
prognosis in, 571
Thyroid, dysfunctions of, 607
in etiology, 114
Timothy-grass bacilli, 30
Tobacco, use of, 682
Tongue, tuberculous ulcers of, 573
Tonsillitis and tuberculosis, 587
Tonsils, infection through, 55
Toxins, hypersensitiveness to, 37, 390, 763
tuberculous, 36
Trachea, displacement of, 410
Tracheobronchial adenopathy, 454
cough in, 460
diagnosis of, 461
emaciation in, 458
fever in, 458
prognosis in, 471
roentgenography in, 468
treatment of, 851
tuberculin diagnosis in, 470
Tracheophony, 466
Traumatic tuberculosis, 638
after surgical operations, 642
clinical aspects of, 640
expert testimony in, 643
pulmonary, 639
tubercle bacilli in, 641
Treatment of abortive tuberculosis, 844
of acute forms of tuberculosis, 849
of advanced phthisis, 845
of arrested cases, 848
climatic, 710
cost of, 711
economic aspects of, 710
effects of, 712
of febrile patients, 777
vs. open air treatment, 697
of complications, 856
of convalescents, 848

Treatment of dietetic, 733
of children, 852
of fibroid phthisis, 850
of incipient phthisis, 844
indications for, 652
individualization in, 729
institutional, 723
medicinal, 746
in advanced phthisis, 846
of cough, 773
of hemoptysis, 783
open-air, 696
abuses of, 705
contraindications, 707
operative, 794
pneumothorax, 794
psychic influences in, 656
specific, 761
suggestion in, 656
symptomatic, 771
tuberculin, 761
of tuberculosis in aged, 854
in children, 850
during the menopause, 855
Tubercle, anatomical, 160
calcification of, 165
caseation of, 164
fate of, 166
healing of, 178
histogenesis of, 164
necrosis of, 165
sclerosis of, 166
softening of, 165
structure of, 160
Tuberculides, 45, 270
Tuberculin, 36
action of, 36, 763
antibodies in, 37
chemistry of, 36
clinical effects of, 390, 766
dangers of, 770
diagnostic value of, 391
dilutions of, 766
dosage of, 391, 762, 766
hypersensitiveness to, 37, 389
preparation of, 36, 766
reaction of, 37, 385, 769
bacteremia after, 391, 770
cutaneous, 386
focal, 385, 390
general, 385, 390
local, 385
as a specific remedy, 761
specificity of, 38, 387, 761
tests, 37
in adults, 72
in children, 72, 387, 470
clinical value of, 391
conjunctival, 389
diagnostic value of, 391
in infants, 452
specificity of, 387, 390
subcutaneous, 389
treatment, 761
administration, 766
Tuberculin treatment in children, 854
dangers from, 769
dilution in, 766
dosage, 766
evidence of inefficiency, 766
hemoptysis during, 720
inefficacy in animals, 764
lack of statistics of, 766
psychic effects of, 657, 768
reaction during, 769
tolerance of, 763
Tuberculolysins, 37
Tuberculosis verrucosa cutis, 585
Tuberculotoxins, 35
Typhoid fever in etiology, 128
tuberculosis and, 598

U

Ulcers, tuberculous, of intestines, 181, 262
of mucous membranes, 573
of stomach, 261
Urban life, tuberculosis and, 77, 97
Uremia, 288, 572
Urinary system, 285
Urine, tubercle bacilli in, 285
antigens in, 393
Urochromogen reaction, 629
Urogenital tract, tuberculosis in, 572

V

Vaccination and tuberculosis, 595
Vas deferens, tuberculosis in, 587
Veins, enlarged, on chest, 301
in children, 464
thrombosis of, 569
Vagotonia, 259
Venesection in hemoptysis, 786
in pneumothorax, 859
Ventilation, 698
Virgin soil, 152
Vocal fremitus, 312
in pleural effusions, 494
Voice sounds, 352
Vomiting, 198
after cough, 197

W

Wages, tuberculosis and, 82, 89, 98, 136
War, effects of, 82, 89
primitive races in, 74
Weight of children, 458
of patients, 265
seasonal influences on, 265
Whispered voice, 352
in early phthisis, 381
Williams's tracheal tone, 405
Wintrich's phenomenon, 359

Z

Zomotherapy, 738

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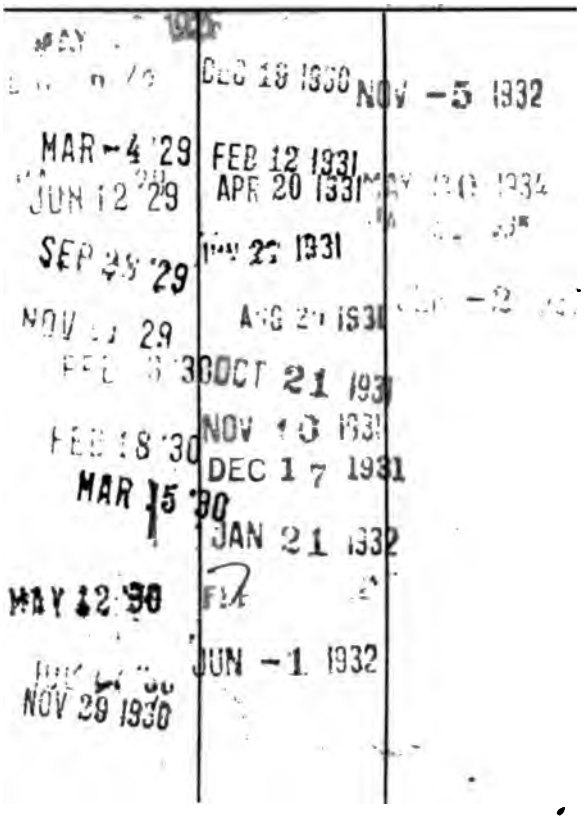
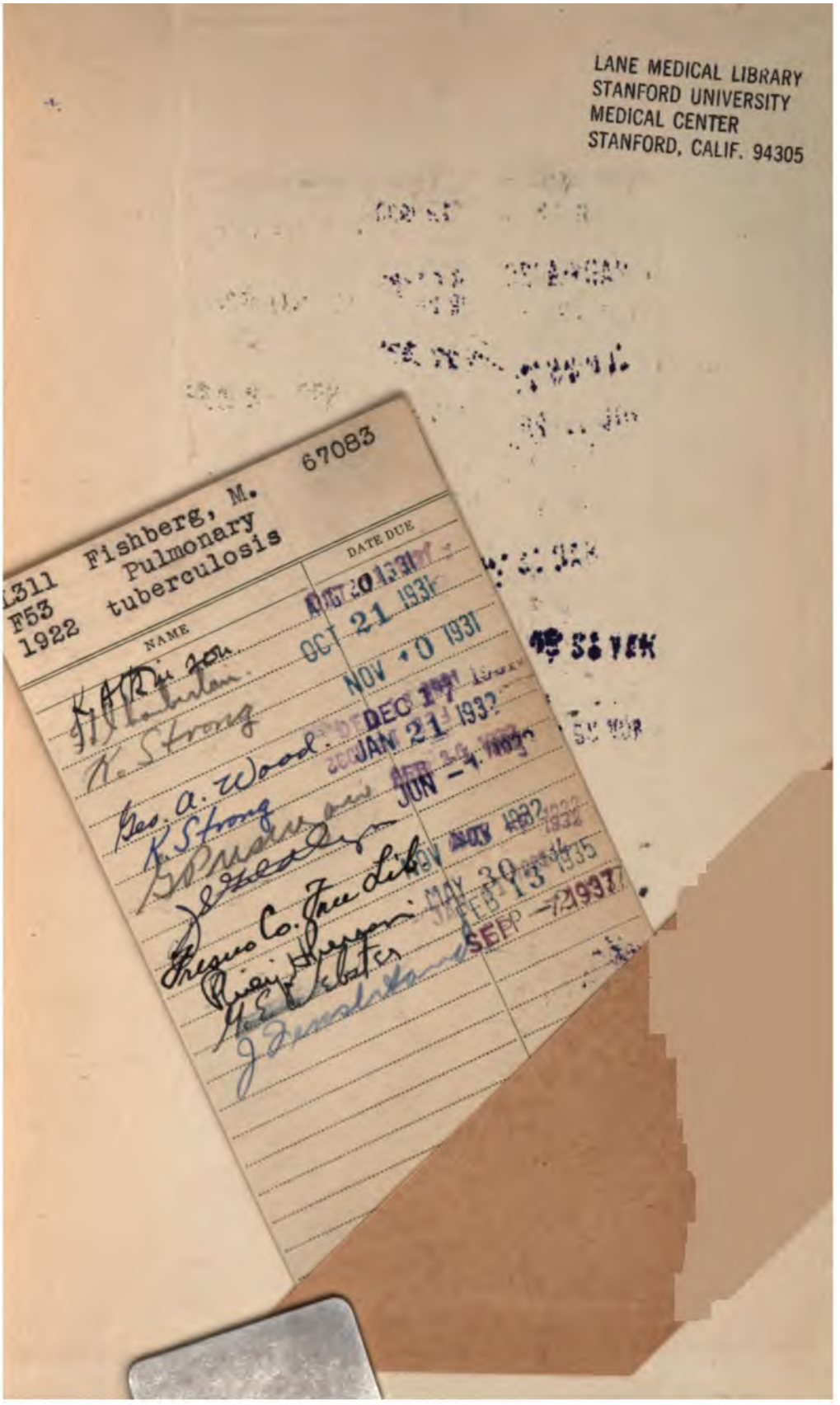
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